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A. L. Scharen, Editor

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PREFACE

One conclusion of the Septoria Diseases of Wheat Workshop held in Georgia in 1976 was to have subsequent workshops of a similar character. That idea was discussed among Septoria workers, from time to time, but didn't really gel until October, 1981, when Lloyd Nelson, Zahir Eyal and Al Scharen were together as consultants to the Brazilian Ministry of Agriculture at Passo Fundo, Rio Grande do Sul. During that period it was agreed that inquiries would be made among workers who had attended the Georgia Workshop as to interest in a 1983 meeting in Montana. The responses were mainly positive, so planning was begun. Joint sponsorship was organized to include USDA, ARS, the Montana Agricultural Experiment Station and the National Wheat Improvement Committee. Barry Cunfer agreed to organize the program and Joe Krupinsky took responsibility for the exhibits and demonstrations.

The response was tremendous! When the workshop opened in Bozeman on August 1, 1983, 85 participants from 21 countries were in attendance. Activities extended over a six day period, with the formal part of the program utilizing three days.

Summaries of activities and presentations are included in these Proceedings. Exchanges of information and research methods should continue to stimulate work on these important cereal diseases. A preliminary discussion of plans for the next workshop, perhaps to be held in 1988, has taken place.

My thanks to all of the people who helped make the workshop a success - secretaries, students, technicians, colleagues, family. It was a great experience. (A. L. Scharen, Editor)

A NOTE ON NOMENCLATURE

Because of the current confusion in the literature, and amongst workers, regarding the nomenclature of the organisms within the complex of diseases known as the septoria diseases of cereals, a working party was convened during the International Workshop on the Septoria diseases of cereals held at the Montana State University Bozeman, August 1 to 4, 1983. The purpose of the working party was to present to the meeting guidelines on the correct useage of both taxonomic and common names for the organisms within this complex.

The working party suggested to the meeting that (1) the correct taxonomic name for these fungi should follow the principles set down in article 59 of the International Code of Botanical nomenclature, and (2) that the common names for the diseases should be based on the imperfect state names of the organisms, which are being used loosely in this context.

A motion was passed at the meeting that, "The taxonomic names of the fungi involved in the septoria disease complex would be based on their perfect state, namely Leptosphaeria nodorum E. Müller, L. avenaria Weber, f. sp. triticea T. Johnson, and Mycosphaerella graminicola (Fuckel) Schroeter, and that the common names of the diseases be septoria nodorum blotch, septoria avenae blotch, and septoria tritici blotch. The lower case "s" will be used for septoria, and septoria nodorum etc. would not be written in italics."

Some of the papers in this proceedings follow the new guidelines; however, others do not since they were prepared far in advance of the workshop. We hope that readers will accept and use the new guidelines in future writings about the septoria diseases of cereals.

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Numbers in parentheses refer to references at the end of each paper. References, figures, and tables are reproduced essentially as they were supplied by the author(s) of each paper.

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PARTIAL RESISTANCE, CULTIVAR MIXTURE, AND EPIDEMIC DEVELOPMENT IN THE SEPTORIA NODORUM - WHEAT ASSOCIATION

D. Gareth Jones[1]

Twenty years ago, there was little research interest in Septoria diseases of cereals. The situation has changed since then with a regular publication of research papers from almost all the wheat growing areas of the world. Pathologists and plant breeders had previously focused their attention on other diseases such as the rusts and powdery mildew; however, a bad Septoria year concentrates the mind on splash-dispersed pathogens, and Septoria diseases are presently very much established in the current list of economically important diseases. Surprisingly, many of the early reports failed to specify the particular species, a surprising omission in light of the fact that countries differ considerably in terms of the dominant Septoria pathogen. In the United Kingdom and West Germany, S. nodorum Berk., the causal organism of glume blotch, is the most prominent and damaging whereas only S. tritici Rob. ex. Desm., the causal organism of leaf blotch of wheat, is of major importance in New Zealand and Tunisia. This lack of distinction is even more surprising when it is appreciated that these pathogens have different sexual forms.

One thing is very clear, both pathogens can be very pernicious and capable of reducing yields by as much as 30 to 40% (table 1). To expand a little on the U.K. situation, S. nodorum has, on several occasions since 1965, been ranked as the most damaging pathogen of winter wheat, but, on average, it comes second to Erysiphe graminis with national losses of between 1 and 7% annually. These figures clearly indicate that the research effort now directed at these diseases is totally justified.

The purpose of this keynote lecture is to provide the background scenario to the subject, possibly to be a little provocative but certainly, or hopefully to create and stimulate an atmosphere of scientific debate. In 40 minutes, it is impossible to be all things to all people, but, by selecting the topic of partial resistance linked to its utilization in terms of disease control, I hope to introduce several research areas of current interest which will be developed more fully in my oral presentation and, no doubt, during this meeting.

Over the past few years, there has been an increasing awareness of the components of partial resistance to disease, not simply as resistance mechanisms but in terms of their effect upon epidemic development. For a predictive evaluation of a host genotype's likely performance in the field, conventional disease assessment of individual plants must be replaced by measurements of resistance components which are known to influence the development of an epidemic in the field. This is not to denigrate the measurement of lesion size and leaf damage which, over a time scale, reflect the severity of the epidemic and the resistance of the host. However, without measurements of such criteria as latent period or sporulation, the assessment is reduced to a retrospective rather than predictive role with its obvious limitations. We would all accept that a more detailed understanding of the individual components of resistance is desirable for the plant breeder but, the extent to which this information may be applied and the epidemiological consequences appreciated and manipulated by the pathologist, the agronomist, or the farmer, is only now beginning to be realized.

The concept of partial resistance is no new phenomenon and, thanks to the reviewers (9, 14) we should all be aware of its characteristics. The generally accepted definition of partial resistance is that it comprises several components, each of which operate at and affect different stages of the epidemic cycle. It is the combination of these components which confers the resistance observed in the field (4). Differences in the field resistance of cultivars are the results of perhaps small variations in the expression of these components accumulated over several pathogen generations. In these days of models and computer simulations, it is now possible to predict the course of epidemics resulting from a multitude of combinations of levels of efficiency of the individual resistance components. The results are most revealing and must surely shape our future breeding and utilization strategies--a theme to which I will return later.

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Table 1.--Losses due to S. nodorum and S. tritici in a selection of countries (Estimates from many fungicidal control comparisons).

Countries	S. nodorum	S. tritici
	Percent	Percent
West Germany	25-30	-
Brazil	-	50
United Kingdom	35-40	10-15
Romania	10	10-25
Australia	-	19

Such is the background to resistance to S. nodorum. As far back as 1968, Bronnimann stated that there were no distinguishable races of this pathogen and hence, no race-specific resistance existed. The implication is that the resistance is partial (16), but the point must be made that it is difficult to prove that such partial resistance is race nonspecific because proof depends upon large numbers of cultivars being tested against large numbers of pathogen isolates. In addition, great care must be taken over the conduct of the tests, it being imperative that the environmental factors and inoculation techniques are standardized.

Brevity demands that the number of questions that can be considered in this paper is very small. The first of these is fundamental to the commencement of a comprehensive resistance screening program and should provide information basic to any subsequent more searching inquiries: "Can we predict field resistance from laboratory measurements of partial resistance components?"

Before this question is considered, it must be realized that precise assessment of field resistance can only be carried out in large scale field trials and under differing environments. These demands immediately put such an exercise beyond the scope of the individual researcher who has, in consequence, to use some other method of evaluation. In the U.K., the National Institute of Agricultural Botany publishes a list of recommended cereal cultivars in which each cultivar is rated for yield and other agronomic attributes along with a value for resistance to particular diseases on 0 to 9 scales; the higher the score, the better the expression of the character. The various ratings are derived from data collected at numerous centers by many observers of varying levels of expertise. At best, they probably only provide a useful guide to the best and the worst. The limitations to the use of such values in statistical analyses are only too apparent, but they are unavoidable to the researcher with scant resources.

The first experiment was designed to compare data of the components of partial resistance obtained from laboratory experiments with the published N.I.A.B. disease ratings for S. nodorum. The results summarized in table 2 show significant variations in all the components of resistance measured, but mostly there were no significant correlations with N.I.A.B. ratings.

I don't propose to elaborate on the experimental techniques used in this experiment; however, briefly, mature, pot-grown plants were inoculated by the placement of measured droplets of spore suspensions on leaves held horizontally by taping onto plates of glass.

Surprisingly, but not without significance, there was no correlation between lesion size and National Institute of Agricultural Botany (NIAB) ratings although there were significant differences between the cultivars. Infection frequency was scored on several occasions and, while we conclude that it could not be used as a very reliable predictor of field resistance, it was interesting that the most susceptible cultivar (cv. 'Maris Ranger') consistently had the highest infection scores. Incubation period gave a significant correlation with NIAB ratings with a range of 6 days between cultivars. Latent period ranged from 21 to 28 days and, while cv. Maris Ranger did have the shortest period, there was no correlation with field resistance rating. The precise measurement of sporulation is most difficult and has been studied using many methods (3, 12). Our method involved placing infected leaf samples in water with an added surfactant, shaking for 2 hours, and then counting spores using a haemacytometer. Over three harvests covering a 14-day period, only the third harvest gave a significant correlation with NIAB ratings.

This was a very simple investigation to evaluate the predictive efficiency of each component. We concluded that there was considerable variation for each component but that no single component could be used as a single, reliable test for field resistance although incubation period and sporulation would be useful in an initial screen. However, logic directs that sporulation is too complex a character to enable prediction of field resistance from a single spore harvest. I would suggest that some combined measure of total sporulation and infectious period, what might be called the "sporulation profile," might give a stronger relationship.

Table 2.--Range of variation in components of partial resistance in 7 winter wheat cultivars.

Component	Range	Correlation with NIAB rating 'r'
Infection frequency	(67 - 97% Day 12)	N.S.
Incubation period	(2 - 8 days)	0.86; P < 5%
Latent period	(21 - 28 days)	N.S.
Lesion area	(2.5 - 13.6 mm^2)	N.S.
Sporulation H1	($7x10^4$-$15x10^4$ spores/ml^{-1})	N.S.
H2	($2x10^3$-$17x10^4$ spores/ml^{-1})	N.S.
H3	($5x10^4$-$18x10^4$ spores/ml^{-1})	-0.86; P < 5%

In order to test the reliability of our test methods, we constructed a "resistance index" by a simple combination of the ranking of the cultivars for each measured component of partial resistance. The results are shown in figure 1, and the very high correlation with NIAB disease ratings confirms that field resistance is the combined expression of the individual components.

In brief summary, the results of the measurement of partial resistance components using the technique described provide some indication of likely field performance but could not be considered as being of precise predictive value. The composite "resistance index" indicates that our measurements could not have been too wayward, and the question now suggested is whether more accurate predictive tests can be devised for individual components? One possibility is the production of more detailed cutltivar "sporulation profiles." Another is the bioassay of amounts of fungal mycelium present in lesions. Some attempts in this direction have given promising results albeit with time-consuming analyses of chitin presence (10). In this context, we have recently developed a more efficient technique involving an ergosterol assay (5). This method is very sensitive and offers a high level of reproducibility.

The second investigation was again concerned with partial resistance components and is a more sophisticated but logical extension of the first investigation. Zadoks (15) had previously shown

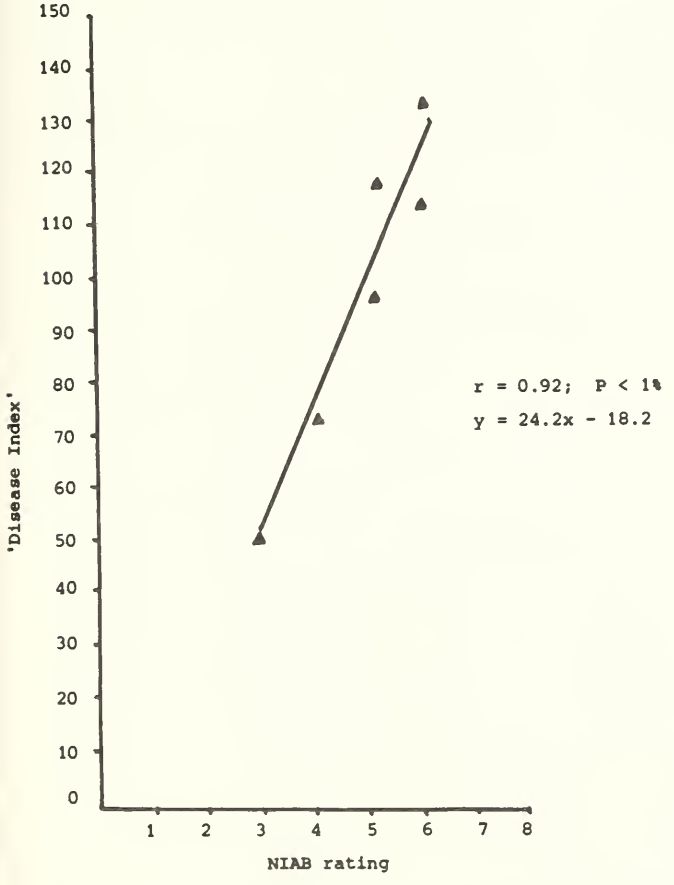


Fig. 1. The relationships between 'Disease Index' and NIAB rating in six winter wheat cultivars.

that by varying one component of resistance at a time in a computer simulation, the progress of the epidemic, as measured by "r" (14) was affected most by latent period, less by a combination of infection frequency and spore production, and least by infectious period. My colleague, Peter Lancashire, has written a computer program to similarly evaluate components of resistance and has included various refinements in terms of epidemiological weightings. Figure 2 illustrates how an epidemic might differ in three cultivars differing only in terms of sporulation and latent period.

The differences are most revealing, but the practical relevance of such differences depends upon the necessary variation being present in the host system and its ability to be manipulated by plant breeding.

Analytical equations may also be used to similarly compare the effects of components of partial resistance. These were used in the second investigation in which the main question asked was: "Do the components of partial resistance act independently of one another in a way that might result in cultivars with very different components having similar levels of field resistance?"

The aims of this experiment were simple, but I hope to demonstrate several new approaches to the analysis and interpretation of the results. For this investigation, 10 winter wheat cultivars were selected all of which had an NIAB rating of either 5 or 6. Again, the experiment was conducted on pot-grown plants in the glasshouse, the second leaf (below the flag leaf) on each fertile tiller, being inoculated with measured droplets of spore suspension. The following components were scored: infection frequency, latent period, lesion area, lesion shape, and sporulation. From these measurements taken at intervals, the rate of growth of lesion area, the rate of change of lesion shape, and the rate of increase of sporulation per unit area were also calculated. Latent period was estimated by probit analysis and was taken as the time by which 50% of visible lesions had sporulated as estimated by the regression line of sporulating lesions against the logarithm of days after inoculation. Lesion area was calculated from measurements of length and width, but, in this investigation, a shape factor was also calculated, which we considered to be more biologically meaningful. Shape factor is expressed as the ratio of lesion length to width with a circular lesion having a shape factor of 1. The larger the shape factor, the more elongated the lesion (along the leaf axis).

A multivariate analysis of variance showed that there were significant differences between cultivars in terms of their components ($P < 5\%$). The results are summarized in table 3, and it can be seen that infection frequencies, lesion areas, and rates of growth of lesion areas were all significantly different, and the rate of change of shape factors was positive, indicating that lesions grew faster parallel to the leaf axis. However, cultivars did not differ in the rate of change of the shape factor. Cultivars differed in the mean density of sporulation ($P < 5\%$) with a range of

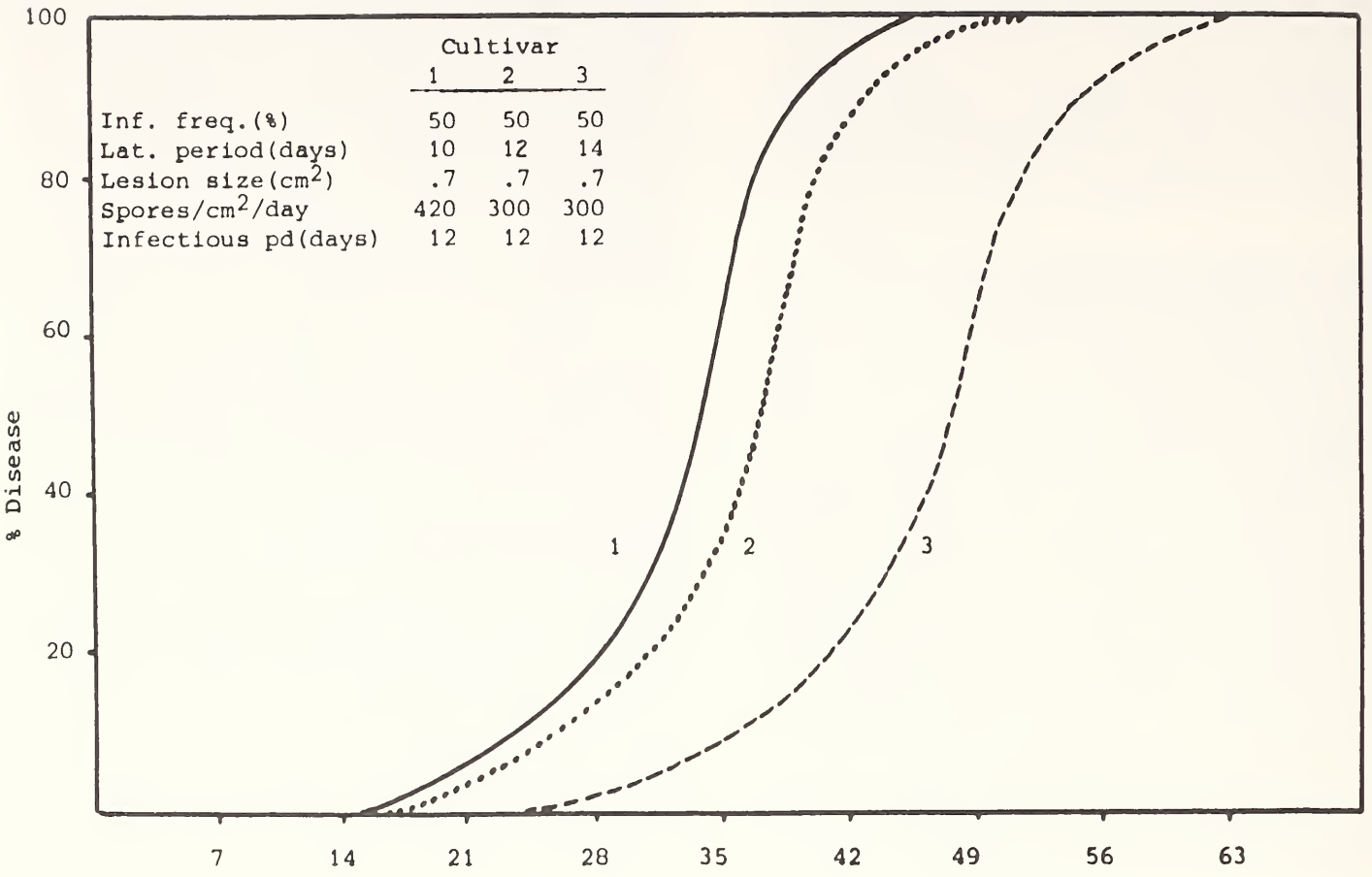


Fig. 2. Plant disease simulation.

Table 3.--Components of partial resistance in 10 winter wheat cultivars (values in parentheses show rates of increase).

Cultivar	Infection frequency	Latent period	Lesion area	Shape factor	Sporulation
	Percent	Days	mm^2		10^3 spores/ml^{-1}
Armada	2.2	22.4	2.7(0.3)	2.8(0.2)	5.6(1.5)
Atou	1.8	24.1	3.1(0.3)	2.8(0.2)	6.4(4.5)
Bounty	2.9	29.7	3.1(0.2)	3.3(0.2)	4.2(6.4)
Brigand	1.2	19.1	4.0(0.4)	4.1(0.2)	6.3(4.0)
Flanders	1.1	23.9	2.6(0.3)	3.1(0.2)	5.0(1.2)
Flinor	1.4	24.7	1.9(0.2)	2.7(0.2)	3.4(1.9)
Hobbit	1.2	20.6	2.8(0.3)	2.8(0.3)	10.2(0.7)
Hustler	1.4	22.0	3.8(0.3)	3.2(0.1)	9.7(2.8)
Freeman	2.4	25.5	2.5(0.2)	3.4(0.3)	4.6(1.5)
Virtue	1.0	24.9	3.2(0.4)	3.3(0.2)	3.8(2.1)
	P < 5%	P < 0.1%	P < 0.1% (P < 1.0%)	N.S. (N.S.)	P < 5% (N.S.)

3400 mm^{-2} to 10,210 mm^{-2}, but the rate of change of sporulation density did not differ significantly between the cultivars.

Probit analysis of the latent period data showed that they more closely fitted a log-normal distribution rather than a normal distribution. The mean latent period for all cultivars was estimated at 23.6 days with a range of 19.1 to 29.7 days, the difference between cultivars being significant ($P < 1\%$).

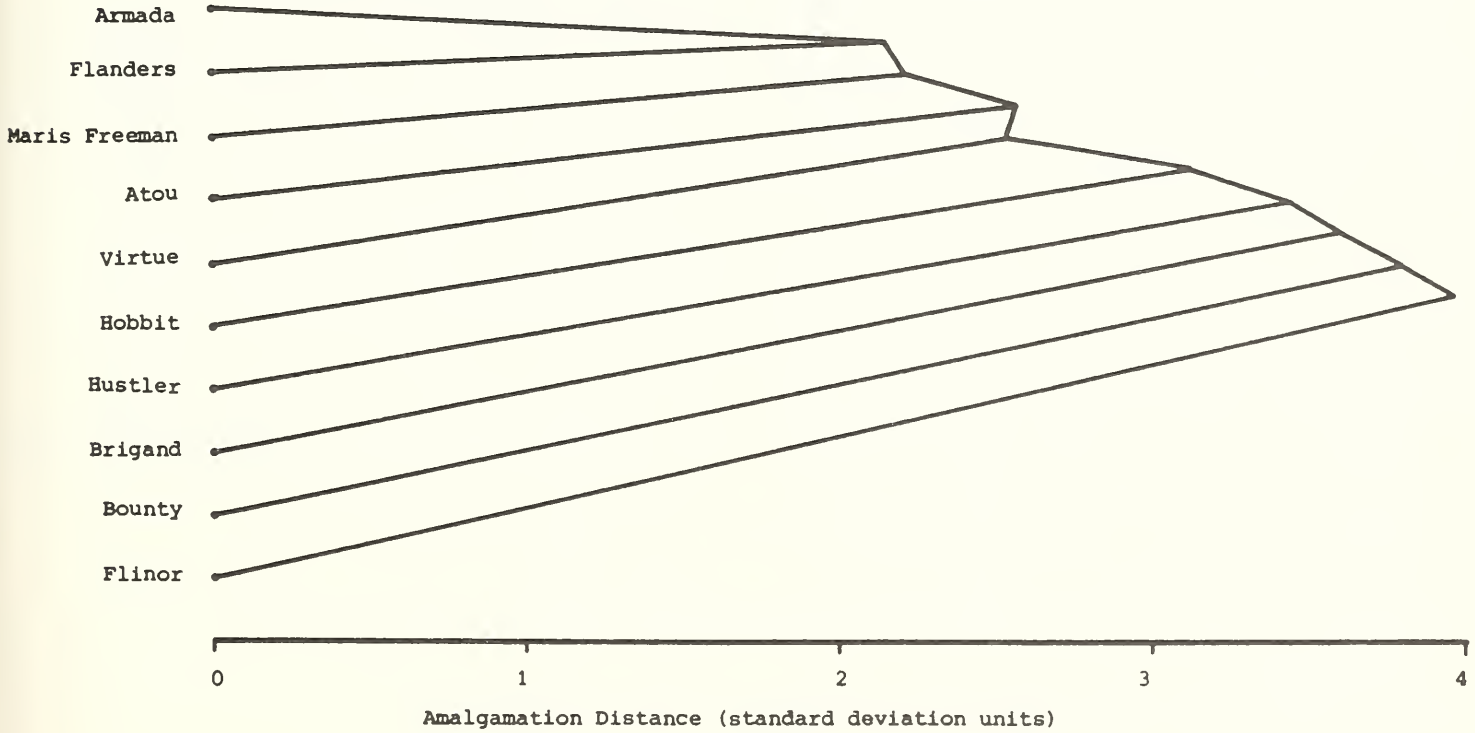
Cluster analysis is a technique for aiding the interpretation of relationships amongst a group of individuals, in this case, cultivars of winter wheat. Cluster analysis has been used extensively in taxonomy and has also been used for classifying plant disease epidemics (8, 13), but there have been no reports of its use for the classification of cultivars according to their components of partial resistance. There are two distinct parts to cluster analysis: (1) measuring the similarity between individuals and (2) linking similar individuals into groups.

The results of the analysis are plotted as a dendrogram (fig. 3). Each node represents the linkage of the two cultivars or clusters specified by the two branches of the tree joined at the node. The distance (in standard deviation units) at which the linkage occurred is shown by the amalgamation distance on the horizontal axis. The interpretation of the results is somewhat subjective, but the pattern clearly shows that there are no distinct groups of cultivars. The diagram indicates that cv. 'Flinor' is least similar to the others, followed by cv. 'Bounty', although these do not form distinct groups.

The same variables were used for correlation analysis as were used for cluster analysis. Bartlett's test of sphericity (2) was used to test whether the resistance components derive from a multivariate normal population. This was found to be the case ($P < 5\%$; 15 DF), and thus it was concluded that the components were independent of each other. Of the significant correlations, latent period was generally negatively correlated with other components as would be expected if it were part of a general resistance trait. However, it was positively correlated with infection frequency and rate of increase in sporulation, which is difficult to explain and is contrary to the results of Shearer and Zadoks (11).

These data gave further indication of the combination effect of partial resistance components and, as in the first experiment, we sought to demonstrate this more precisely in the form of a mathematical model which would describe the overall cultivar resistance. For this purpose, my colleague, Peter Lancashire, produced what we now call the r-index. It has the important difference from the conventional r of Van der Plank of being calculated using data of the individual partial resistance components. In essence, it is the reciprocal of the conventional r, and, as such, a low value of the r-index indicates a rapidly progressing epidemic and hence low resistance. It is not directly comparable to values of r determined from field experiments because the values of the dispersal

Fig. 3. Cluster analysis dendrogram of 10 winter wheat cultivars classified by eight components of partial resistance to S. nodorum. The classification variables are standardised infection frequency, latent period, lesion area, shape factor, sporulation density and rates of increase of lesion area, shape factor and sporulation density.



and viability parameters in the field are not known. However, although not intended to be a precise indicator of field performance, the *r*-index may be a useful test for comparing cultivars. The model for the *r*-index does not take into consideration environmental interactions. In a more refined model, these factors would be taken into account albeit at the expense of greater complexity and more data collection. The values for the *r*-index for the 10 cultivars are shown in table 4. The coefficient of variation was only 5.6% thus, despite the significant multivariate differences in the components of resistance between the cultivars, when combined together in the *r*-index they appear very similar. This implies that the cultivars would have very similar field resistances and is in accord with their published NIAB ratings.

As a double check on the validity of the *r*-index, two theoretical "cultivars" were constructed using the most "resistant" and most "susceptible" components from the 10 commercial cultivars. The *r*-indices for these synthetic cultivars are also shown in table 4. The values (susceptible = 5.2; resistant = 23.1) fall outside the 5% confidence limits for individual values of the real cultivars.

These results give clear instructions to the plant breeder. Given that it should be possible to genetically segregate the components of partial resistance, evidence of their independence having been demonstrated by the Bartlett's test of sphericity, it should be possible first to theoretically design a more resistant cultivar and then to accomplish this in practice. The gauntlet is now thrown at the feet of the breeders.

To conclude this paper, I would now like to briefly consider the use of partial resistance in cultivar mixtures. Mixtures are being used commercially in the United Kingdom and, although our initial publications indicated considerable potential for disease control by this approach, more recent work suggests there are limitations and that a degree of caution should be exercised.

Much of the evidence of mixture benefits, as with other diversification schemes, originates from work with airborne, highly specialized pathogens, such as rusts and powdery mildews. However, over the past few years, my colleagues and I at Aberysteryth have been studying and evaluating mixtures as a means of controlling splash-dispersed pathogens, in particular *S. nodorum* and *Rhynchosporium secalis*. We have published a model to predict the likely outcome of disease in mixtures of cultivars of differing combinations of efficiency levels in the components of partial resistance.

The model (6) utilizes the values of the various components and predicts the likely level of disease in the mixture compared with the means of the constituent cultivars in pure stands and the estimated arithmetic means of the cultivars in varying mixture proportions. A typical prediction, based on data from the first experiment described in this paper, for various cultivar proportions in a binary mixture is shown in figure 4b. The mixture advantage is very obvious.

The whole concept of mixture benefit is based on achieving the most efficient combination of components of partial resistance in the mixture cultivars. Theoretically at least (fig. 4a), the model also predicts that it is possible to exacerbate the disease levels if the cultivar components are incorrectly matched. This effect is shown by the dotted line and whilst this has still to be tested in practice--and we don't know if such cultivars exist--it does strongly emphasize that caution should be taken before generally recommending the use of mixtures and that even greater care be exercised in the selection of cultivar combinations for use in commercial mixtures.

Evidence that the model works and that mixture advantage accrued has been furnished by a field experiment using mixtures of two spring wheat cultivars, the susceptible cv. Kolibri (NIAB 3) and the moderately resistant cv. Maris Butler (NIAB 6) (6). The results are shown in figure 1c where it can be seen that the mixture advantage was very apparent in treatments 2, 3, and 4 in

Table 4.--Calculated 'r-indices' for winter wheat cultivars and 2 theoretical cultivars (values in parentheses show 1979 NIAB disease ratings).

Cultivar		'r-index'
Armada	(6)	11.3
Atou	(5)	11.6
Bounty	(5)	13.4
Brigand	(6)	10.3
Flanders	(6)	15.8
Flinor	(5)	19.6
Hobbit	(5)	10.3
Hustler	(6)	9.4
Maris Freeman	(5)	13.3
Virtue	(6)	16.5
'Susceptible'		5.2
'Resistant'		23.1

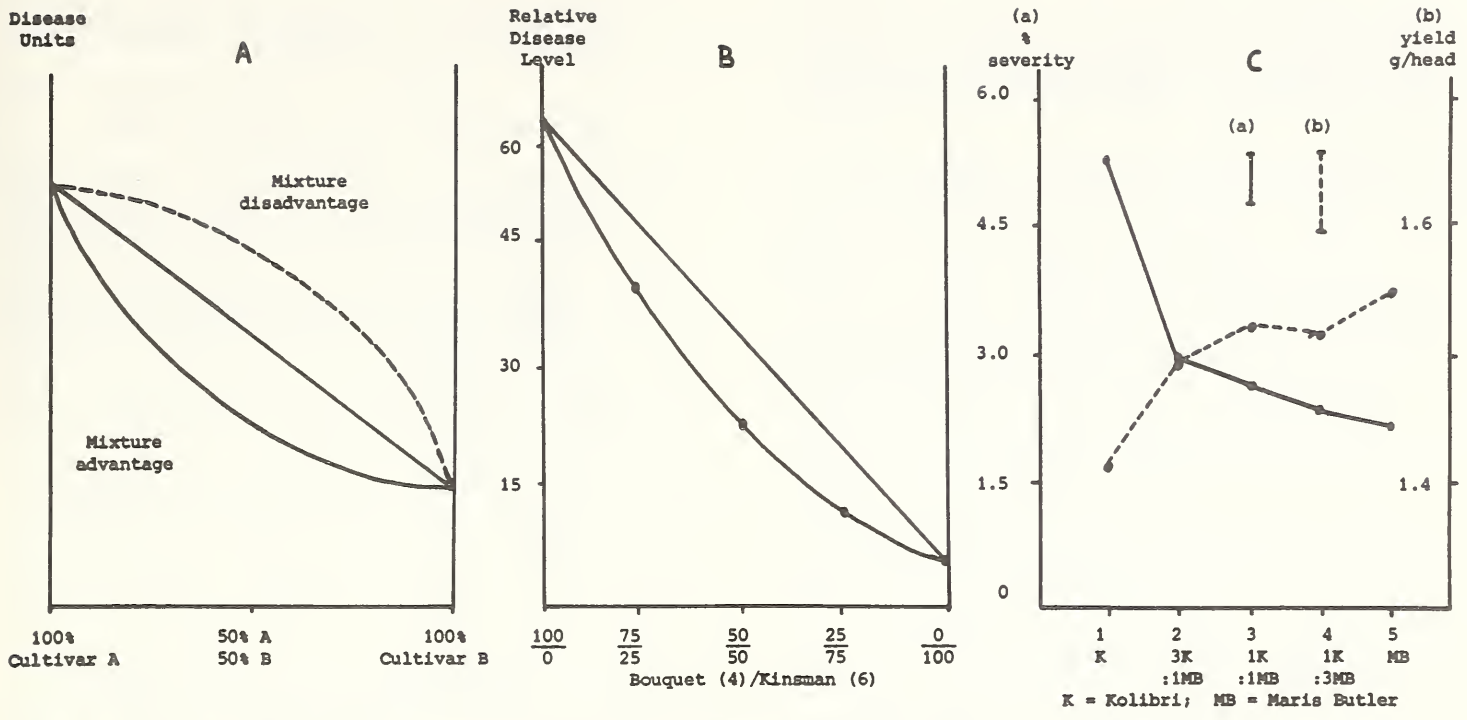


Fig. 4. A = Model predictions; B = Predictions from experimental data; C = Field data.

terms of depressing disease to a significantly lower level than that predicted from the means of the pure stands. The yield data as expressed as g/head, reflected this reduction in disease, but the differences failed to reach statistical significance. In discussing these results, it was stated that, in view of the overall disease levels in that particular experiment being rather low, large effects on yield were not to be expected and that we were of the opinion that the results reflected genuine mixture advantages.

However, since those first experiments, we have now tested the model to exhaustion, that is to the point when there is so much disease that there is very little plant tissue to infect and there is an abundance of inoculum. Surprisingly, the model then predicts the disappearance of the mixture advantage that was apparent at low disease levels. This result necessitates a reappraisal of our whole philosophy on the advantages of mixtures as it seems quite likely that, under severe epidemics, no advantage would be predicted with certain cultivar combinations. It also might explain the negative results of other workers using various mixture combinations.

Finally, may I suggest that the most precise approach to the prediction and evaluation of disease resistance is the measurement of individual components of partial resistance coupled with detailed knowledge of their significance in epidemiological terms. Such studies should endeavor to incorporate measurement of the component/environment interaction, and these would include soil factors, fertilizer application, and the effect of fungicides. I hope I have shown that the use of simple and sophisticated models, coupled with the ability of the computer to assimilate the generated data, will allow the study of the variation of individual components of resistance in different host backgrounds under varying cultural regions. The use of such simulations will not only facilitate the theoretical design of new resistant cultivars, which would serve as the blueprints for the plant breeder, but also will facilitate the prediction of the outcome of permutations of cultivar mixtures in terms of disease progress.

ACKNOWLEDGMENTS

The views expressed in this paper are entirely my own, but they are based upon joint work with my previous graduate students, Helen Griffiths, Mike Jeger, and Peter Lancashire, whose efforts, along with those of my collegue, Ellis Griffiths, I gratefully acknowledge.

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GENETICS OF HOST-PATHOGEN INTERACTIONS TO THE SEPTORIA SPECIES OF WHEAT

A. A. Rosielle and W. J. R. Boyd[1]

The genetics of host-pathogen interactions to the Septoria spp. of wheat is of concern to plant breeders and pathologists faced with the task of developing resistant cultivars. Information on this topic is relatively sparse; in fact, it was only 12 years ago in the review by Shipton et al. (29) that we concluded for Septoria nordorum that "true resistance has not been shown to exist," and for S. tritici that "much of the data in literature on resistant varieties may be of questionable value." The past 12 years have seen an increased research effort resulting in a better understanding of sources of resistance, the genetics of resistance, and host cultivar-pathogen isolate interactions to these diseases. We will draw attention to this progress in this paper.

We will concentrate on the two major Septoria diseases of wheat and consider them separately. It is important to remember that these diseases are, in fact, separate diseases. The perfect stage of Septoria nodorum (Berk.) is Leptosphaeria nodorum (Muller), while that of Septoria tritici Rob. ex. Desm. is Mycosphaerella graminocola (Fuckel) Schroeter. The major justification for considering the diseases together is that they produce similar symptoms, and, in some parts of the world, they may occur together in the one crop. However, the two diseases are quite distinct in terms of their host-pathogen interactions as we will discuss herein.

S. NODORUM

Our earlier conclusion (29), that true resistance to S. nodorum has not been shown to exist, is still valid if framed against a conventional background of a clear-cut distinction between resistance and susceptibility. However, numerous papers have been published over the last 10 years showing that variation in host reaction exists in wheat to this disease (2, 11, 19, 25). This variation in host reaction is generally considered to indicate "resistance." However, the resistance is not of a high order, and given a sufficiently favorable environment for the pathogen, most resistant genotypes would be considered susceptible by definitions used for resistance with other diseases.

A single dominant gene for resistance was reported in the winter wheat cultivar 'Atlas 66' by Frecha (6). Kleijer et al. (10) identified this resistance as being predominantly on chromosome 1B. However, Scott and Benedikz (25) expressed doubt whether the resistance in Atlas 66 could, in fact, be attributed to a single gene, and Kleijer et al. (10) failed to reproduce the 3:1 F_2 ratios reported by Frecha (6). Most studies indicate that resistance to S. nodorum is not under control of readily identifiable genes. That a number of genes are sometimes involved in resistance is shown by Scharen and Krupinsky's data (24) of substantial transgressive for resistance in a number of wheat crosses.

Bronnimann (2), Scott and Benedikz (27), and Mullaney et al. (13) have concluded that resistance is predominantly under additive genetic control. However, in addition to additivity, Nelson and Gates (15) found evidence for dominance and additive x additive epistasis. Heritability of resistance based on leaf area damaged appears to be relatively high, of the order of 50 to 60% in plots with two replicates in one study (20). Bronnimann (2) reported that tolerance, as measured by percentage of average individual seed weight in infected to noninfected plots, had a heritability of 65%.

Resistance to S. nodorum has often been correlated to some extent with characteristics such as late maturity, winter growth habit, or tall plant height (3, 20, 27). Given that late maturity and tall plant height are generally undesirable traits in modern wheat cultivars, it is important to consider the extent to which resistance can be identified which is independent of these characteristics. Rosielle and Brown (20) showed that substantial variation among selections from crosses in the trait seed weight percent (equivalent to Bronnimann's [2] tolerance described earlier) could be attributed directly to the extent of flag leaf and head area damaged, independent of heading data and plant height. Scott et al. (27) similarly concluded that considerable resistance exists independently of heading date and plant height. Therefore, the results from both Rosielle and Brown (20) and Scott et al. (27) suggest that correlations between apparent resistance and late heading date or tall plant height are not likely to provide a major impediment to selection for resistance.

How do maturity and plant height affect symptom expression to S. nodorum? In the field, disease "escape" associated with less favorable environments or microenvironments for infection may be partly involved. Yield reductions from the disease occur principally through reduction in individual seed weight and, to a lesser extent, decrease in number of seeds per ear, resulting from infection of the ear and flag leaf (1). In many wheat-growing environments, late-maturing cultivars reach anthesis in a generally drier period than early-maturing cultivars, that is, they may escape serious damage. According to Eyal (3), long dry periods toward the end of the growing season interrupt the pathogen's progress toward the upper plant parts from the lower leaves. In environments or seasons where rainfall is high during or after anthesis, correlations between resistance and maturity would be expected to be less important. Short plant height may favor the pathogen by creating a more conducive microenvironment for infection. It may also assist dispersal of the pathogen from leaf layer to leaf layer by the smaller distance between consecutive leaves.

Disease escape may not be the only factor involved in late maturing cultivars and taller cultivars

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showing increased resistance. We have consistently observed that resistance of artificially inoculated seedlings tends to be higher in later-maturing cultivars. Similarly, resistance in winter wheats tends to be higher than that in spring wheats at the seedling stage (23). There may, therefore, be a pleiotropic effect between resistance and plant maturity independent of escape. Scott and Benedikz (26) suggest that tall plants are inherently more vigorous than shorter plants. Additional research is needed to clarify the relative importance of late maturity and tall plant height on resistance and the mechanisms by which such resistance is achieved.

Very few studies have examined correlations between seedling and adult plant resistances. Unpublished field data from our program in Western Australia indicate that these correlations are often low. Rufty et al. (21) observed a good correlation (0.86) between ranking of cultivars as seedlings in the greenhouse and their rank in the field. However, Scharen and Eyal (23) reported that cultivars which show seedling resistance are sometimes susceptible as adult plants. Correlations between seedling and adult plant resistance in the field in different environments would be expected to be influenced by maturity differences among cultivars.

The low level of resistance in wheat to S. nodorum indicates that physiological specialization and the occurrence of distinct pathogenic races may not be important for this disease. However, evidence for a degree of specialization exists. S. nodorum may occur on both wheat and barley (as well as a number of other grass species). Results from cross inoculation studies show that wheat isolates are more pathogenic on wheat than on barley, and barley isolates are more pathogenic on barley than on wheat (5, 9, 22, 32). However, specialization to either wheat or barley (or other alternative hosts) can be apparently induced by passage through that host (5, 7, 8, 22, 31). The mechanism by which this occurs is not clear. Specialization to different wheat cultivars has also been demonstrated (22, 23, 30). Attempts have not been made to classify isolates into races as the distinctions between them are not large. In view of the rapidity with which changes may be induced by passage through alternative hosts, such classifications may prove futile. However, specialization or induced specialization may be extremely important in breeding for resistance to S. nodorum. Resistance that transcends such specialization or induced specialization is obviously most desirable.

S. TRITICI

Resistance to S. tritici has generally been easier to identify and is of a more distinct nature than resistance to S. nodorum. Indeed, simply inherited resistance to S. tritici has been reported by numerous authors (12, 14, 18, 19). The single dominant gene for resistance in the cv. 'Bulgaria 88' has been used in Indiana in breeding the soft red winter wheat variety 'Oasis' (16). In Western Australia, simply inherited resistances from cvs. 'IAS-20', 'Veranopolis', and 'Seabreeze' (19) have been backcrossed into the susceptible Australian cvs. 'Gamenya' and 'Tincurrin'.

Genetic relationships between the different sources of resistance to S. tritici have not been studied, and it is not clear to what extent either different loci or different alleles may be involved. This information is needed if breeders are to accumulate (pyramid) resistance genes in a single cultivar or to develop multilines. It is not known whether different genes for resistance will act together additively.

The inheritance of tolerance as defined by the ability to produce a high 1,000 kernel weight under epidemic conditions was studied by Ziv et al. (33). They concluded that a small number of additive loci may be involved in some crosses while in others the inheritance is more complex.

As with S. nodorum, resistance to S. tritici has been associated with late maturity and tall plant height (3, 19, 28). In the field, plant height and late maturity may act in the same way as for S. nodorum, that is, they may lead to disease escape associated with less favorable environmental or microenvironmental conditions for infection and dispersal. However, Rosielle and Brown (19) found that short, early maturity, resistant lines could be selected within crosses, suggesting that these associations may be at least partially due to linkage. The CIMMYT spring wheat improvement program has also been very successful in developing cultivars which are resistant, short, and early maturing.

Of major concern is the use of single-gene resistances to S. tritici is the possibility that physiological specialization exists. Resistance to S. tritici is more definite than that to S. nodorum, and the simply inherited nature of much of the resistance suggests that it may be short lived in a commercial cultivar. Evidence for physiological specialization in S. tritici exists (4, 17). Eyal et al.(4) showed that specialization existed in Israel, particularly between isolates from bread wheat and those from durum wheat. Hann and Griffiths (7) observed a decrease in virulence on wheat of a wheat isolate when it was passed once through various alternate parts. The decrease in virulence differed for different alternate hosts.

FUTURE RESEARCH NEEDS

Considerable progress has been made in our understanding of the genetics of host-pathogen interactions to the Septoria diseases of wheat over the past 10 years. However, deficiencies exist in our knowledge, and breeding for resistance is by no means a routine procedure. Without implying priorities, we suggest that the following areas are in need of increased attention.

a) The extent to which cultivar maturity and plant height may influence host reaction independent of disease escape factors, the mechanisms behind these responses, and the extent to which pleiotropy and linkage are involved.

b) The occurrence and importance of physiological specialization and its implications to resistance breeding. The mechanisms of induced specialization.

c) Recurrent selection as a means of accumulating genes for resistance. Estimates of genetic parameters (additive and dominance genetic variances and genetic correlations between traits) in populations undergoing recurrent selection will be required.

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ROSIELLE - SPEAKER

Q. Hosford: We also have noticed some of these very late maturing wheats are resistant, but they could never mature in North Dakota. Sometimes, when a wheat variety is planted at the normal time and the same wheat variety is planted 2 or 3 weeks later, then the later planting seems to be much freer of leaf spotting.

A. I think Roger Boyd has observed similar phenomena with diseases of barley . . . and I don't really know what the explanation for it is, other than that plant age seems to be associated with susceptibility or resistance.

Comment by Jim Frank: In Pennsylvania, we found that the situation has nothing to do with maturity. It's just strictly the time when you put the plant in the field because young plants at growth stage 3 may be loaded with Septoria nodorum lesions.

HETEROKARYOSIS AND HETEROKARYON INCOMPATIBILITY IN SEPTORIA NODORUM

A. C. Newton and C. E. Caten[1]

INTRODUCTION

Isolates of Septoria species collected from the field differ in both pathogenic and cultural characters (1, 4, 7). It is difficult to conclude anything about the genetic basis of this variation from field collections and studies of single isolates under controlled conditions required for this purpose. Such studies have shown that individual isolates, including ones originating from a single spore, can vary extensively (4, 5, 6, 8). It has been suggested that this variation reflects the segregation of different genotypes from complex heterokaryons arising through the accumulation of mutant nuclei within a mycelium and nuclear exchange between mycelia following hyphal fusion (4, 5, 6). This interpretation may be questioned, however, since heterokaryosis has not been demonstrated in any Septoria species and its occurrence is restricted by genetically determined incompatibility systems in many species of filamentous fungi (2, 10). The work reported here was undertaken to determine whether heterokaryons can be formed in Septoria nodorum and to test for the presence of a heterokaryon (vegetative) incompatibility system.

Confirmation of heterokaryosis requires demonstration of the presence of two or more genetically different nuclei in the same cytoplasm. Natural phenotypic differences between isolates are unsatisfactory for this purpose as they may reflect cytoplasmic rather than nuclear differences and it is necessary to deliberately mark the nuclei with mutations. Auxotrophic mutations are particularly suitable since they serve not only as markers but may also be used to select for heterokaryosis, thus increasing the sensitivity of the test. Such mutations are generally laborious to obtain, particularly if many isolates are involved as in testing populations for heterokaryon incompatibility. We have overcome this problem by using a system which allows positive selection of nitrate nonutilising mutants through their associated resistance to chlorate (3).

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MUTANT ISOLATION

Wild-type strains of S. nodorum grow well on a Czapek-based defined medium (Cz-N) with either sodium nitrate (NO_3), sodium nitrite (NO_2), ammonium tartrate (NH_4), or L-arginine (arg) as nitrogen source. With arginine as nitrogen source and 0.5M potassium chlorate (ClO_3), wild-type strains produce a thin, spreading nitrogen-starved growth. Dense platings of spores onto Cz-N+arg+ClO_3 medium produced a background growth of starved mycelium with occasional vigorously growing colonies. These chlorate-resistant mutants were isolated and purified and then tested for their ability to utilise NH_4, NO_3, NO_2, or adenine as nitrogen sources. On this basis, the mutants fell into three major types (table 1) which correspond to those found in Aspergillus nidulans where they arise through mutations in single chromosomal genes (3). All three mutant types were unable to utilise NO_3 and produced a thin nitrogen-starved mycelium on Cz-N+NO_3. Representatives of these mutant types were obtained in most of 23 strains of S. nodorum isolated from England, Wales, Ireland, and Denmark.

HETEROKARYOSIS AND COMPLEMENTATION

When two mutants of the same strain but of different type were inoculated about 1.75 cm apart on Cz-N+NO_3 medium they produced a band of thick wild-type growth where the thin mycelia met, that is, they complemented each other. Mass hyphal subcultures from this band onto fresh Cz-N+NO_3 medium gave colonies which grew as wild-type. To determine whether these prototrophic mycelia were heterokaryons, revertants, or simple mixtures, single hyphal tip subcultures were made. Of 100 such subcultures from one mycelium, 26 were still prototrophic while 61 had the nutritional characteristics of one parent and 13 of those of the other. Several of the prototrophic subcultures were sporulated and single-spore derivatives established. Only the two mutant parents were recovered in these single-spore progenies.

These results demonstrate that a significant proportion of hyphae in the prototrophic mycelia contained both nuclear types and hence, that these mycelia were heterokaryons. The absense of prototrophs among the single-spore progeny of these heterokaryons indicates that the heterokaryotic state cannot be transmitted through spores, which is consistant with the claim that spore initials

Table 1.--Growth responses of wild-type and chlorate-resistant mutants.

	Growth on Cz-N supplemented with				
Mutant type	NH_4	NO_3	NO_2	Adenine	Arg+ClO_3
Wild type	+	+	+	+	-
nia	+	-	+	+	+
cnx	+	-	+	-	+
nir	+	-	-	+	+

in S. nodorum are uninucleate (9). When two mutants which complemented in the tests described above were separated by a cellophane disk, no complementation was observed, indicating that hyphal contact and probably heterokaryon formation are essential.

HETEROKARYON INCOMPATIBILITY

Complementation between nia and cnx mutants was then used as a criterion to test for heterokaryon formation between independently isolated strains. Of 252 different interstrain combinations, only 4 (1.6%) showed complementation and, hence, were considered heterokaryon compatible. The remaining combinations (98%) were heterokaryon incompatible. On the basis of their ability to form heterokaryons, 23 strains of S. nodorum were classified into 19 heterokaryon compatibility groups. Fifteen of these groups contain only a single strain, while the remaining four each consist of two strains, with the two compatible strains in each case originating from the same locality. However, a common origin does not ensure compatibility since other pairs of strains from the same locality were not compatible.

CONCLUSION

These studies have established that heterokaryons are formed readily between mutants of the same strain of S. nodorum and that these heterokaryons are stable on mass hyphal transfer. Most pairs of unrelated strains failed to form heterokaryons, however, suggesting the presence of a genetically determined incompatibility system comparable with those in other fungi (2, 10). Thus, the contribution of heterokaryosis to the variability of S. nodorum is likely to be restricted to the accumulation of mutant nuclei within a parent mycelium and to interactions between clonally related mycelia. Since single spores are homokaryotic, genetic phenomena other than heterokaryosis must be considered to account for persistent segregation through successive single spore generations.

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CATEN - SPEAKER

Q. Did you have difficulty defining compatability? There seems to be major qualitative differences in terms of amount. Do you put any significance to that?

A. I think we had no trouble ever in classifying a comparison as being obviously compatable or incompatable. They fell into those two major groups. There might be degrees of compatability or some slight degrees of incompatability, but I think there was a major qualitative effect, which is what we picked up and concentrated on. Now, if you had some more refined way of measuring it, you might be able to show that some bounds of complementation were more extensive than others, so you have quantitative components superimposed on both those measures.

Q. That's the impression I got. Is there any confirmation of it?

A. I think the confirmation is basically qualitative and that is true. I mean, similar systems have been described in quite a number of other fungi.

Q. Can you speculate if we can project this hypothetical system to genes for pathogenicity?

A. I'm not quite sure how you want me to project that.

Q. If we could hypothetically follow a gene by itself, measure it when projected between different carriers, a change of genes for pathogenicity might be detected.

A. The first point I made is that as in the other fungi, we have incompatability, and this incompatability system is purely restricted to the vegetative stages. It stops exchange of the nuclei between vegetative cells. It doesn't stop the hybridizing through the normal sexual cycles. There is no evidence that these cannot hybridize sexually. The opportunities for anastomoses are probably going to be pretty rare. We have pretty good evidence that it's going to be restricted in the field.

METHODS USED IN BREEDING FOR RESISTANCE TO *SEPTORIA NODORUM* IN SWITZERLAND

P. M. Fried[1]

In a breeding program, it is advantageous to be able to select in each generation for resistance to *Septoria nodorum*. Because of the erratic occurrence of the disease at our research station, we have to inoculate our breeding material every year. Inoculation can occur at different growth stages and with different spore concentrations. We inoculate the F_2's to F_5's once (500 L/ha spore suspension, 5 x 10^6 spores/ml at the booting stage of the early lines, and if disease onset if very low, a second inoculation after heading of the latest varieties is done at a reduced spore concentration (1 x 10^6 spores/ml). More advanced breeding material of which enough seed available (usually F_6 to F_{15}) is tested in a special Septoria nursery.

Research of several authors (1, 2, 4) has shown that yield reduction caused by *S. nodorum* is highest when inoculation takes place right after heading. From these observations, it was concluded that for maximum differentiation between varieties inoculation should be carried out at that growth stage. In a study to determine the influence of peduncle length on yield reduction, we observed that inoculation after heading can reduce the kernel weight to such an extent that selection on this variable becomes rather difficult (3).

Another factor which interacts significantly with the disease progress is the weather following the inoculation day. If each entry in a nursery is inoculated individually right after heading, the period which is required to do this may last from 2 to 4 weeks under our conditions. A comparison between lines becomes then a comparison between interactions of disease resistance X weather conditions, and very often the late varieties are classified as being the most resistant ones.

Measuring the yield reduction by comparing the yield or a yield component between inoculated and noninoculated plots allows us to identify tolerance to the disease. In our current breeding material, there appears to be no tolerance present since high correlations are detectable between the reduction in the grain weight and the areas under the disease progress curves.

If yield measurements are made, then the control plots should be protected chemically. The reason for this is the following: the yield or yield component of a susceptible cultivar is reduced in the control plot, as well. Calculating the quotient inoculated/control makes the susceptible cultivar appear more resistant than the resistant cultivar.

In addition, cultivars with low kernel weights tend to reduce this component less severely than cultivars with high kernel weights.

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Considering the above factors and taking into account that measuring the yield reduction requires a high amount of labor input, we have been adapting our breeding method continuously during the past 3 years. The method we use now for the most advanced breeding lines (beginning in the F_6) is the following:

Each entry is planted in a six-row plot 1.30 m long. The plots are arranged in such a way that they can be inoculated with regular spraying equipment mounted on a tractor. We inoculate the whole nursery a first time when the earliest varieties are at the late boot stage (500 L/ha spore suspension, spore concentration = 5 x 10^6 spores/ml).

Then, the entries are grouped into three or four maturity classes according to their heading date. At a given day, when about one third of the entries have headed, each entry in maturity group 1 (early lines) is inoculated individually with a hand-sprayer, again with a 500 L spore suspension/ha, but at a reduced concentration (1 x 10^6 spores/ml). Inoculation of the entries in the other maturity groups is done accordingly at later dates. We try to have 25 to 30 percent of all lines in maturity group 1, 40 to 50 percent in maturity group 2, and 25 to 30 percent in maturity group 3.

Four to six disease readings (percent area with necrosis) are then taken at about 7 to 10-day intervals on the whole plant, the flag leaf, and the heads. Assessments begin when the first symptoms become visible. The areas under the disease progress curves (ADPC) are then calculated and tabulated. Comparisons for selection can now be made but only between entries in the same maturity group. However, this does not completely satisfy our objectives since comparisons between all entries are of interest to the plant breeder. In order to achieve this, I tested different ways of transforming the data. At the moment, the following appears to be the most promising:

For each maturity group, the average of the ADPC for the whole plant, the flag leaf, and the heads is calculated. Then, the differences between the means of each group are determined.

Now, one has to determine one group as the reference group (we take the maturity group 2). The values of the other groups are then projected into the reference group by adding (or subtracting) to each value the difference between the means of the reference group to the group to be projected. This then allows us to make comparisons between all entries in the nursery. The assumption is made that the distribution for resistance within each maturity group is the same, which might not always be the case; however, for selection in a breeding program of a certain volume, this would not be of great practical consequence.

In our breeding program, we like to use a scale from 1 to 9 for as many variables as possible, 1 being very resistant, 9 very susceptible. It is now possible to transform the above mentioned ADPC into values from 1 to 9 by calculating the mean of the group and assigning to the values, for example, 1/2 standard deviation(s) around the mean a value

of 5, from - 1/2 s to - 1 1/2 s a 4, from + 1/2 s to + 1 1/2 s a 6, and so on. For a quick screening of the data, this method can be very useful, but it has the known disadvantages of classifying data into groups.

In the future, we will try to increase the number of replications for each variety and to reduce the number of disease readings to those only where large differences between the entries can be seen. We also test if we could induce a more natural Septoria epidemic by inoculating the nurseries more often but at lower spore concentrations, beginning at an earlier growth stage.

FRIED - SPEAKER

Q. Shaner: Do you make minor adjustments in the timing of your inoculations according to the weather?

A. We have rain, which is one big factor, and then holidays. One fourth are planted in the first group, which are the early ones, about one-half are the middle ones, and one-quarter are the late ones. We have problems every year, especially after the holidays!

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THE EFFECT OF RHT-2 AND OTHER HEIGHT GENES ON RESISTANCE TO SEPTORIA NODORUM AND SEPTORIA TRITICI IN WHEAT

P. R. Scott and P. W. Benedikz[1]

Tall cultivars of wheat tend to be more resistant than dwarf cultivars to Septoria nodorum. However, this need not indicate a genetic relationship between height and resistance: for example, it might merely reflect fortuitous susceptibility in the Japanese donors of dwarfing genes.

To test for a genetic association between height and resistance to S. nodorum, random F_3 families were raised from a cross between tall and dwarf cultivars. Thus, genes for height and genes for resistance were allowed to segregate at random. Any tendency to segregate together must then indicate some degree of genetic linkage or pleiotropy. Between 1976 and 1982, height and the incidence of S. nodorum were measured in inoculated field trials (0.1 m^2 plots) for groups of F_3 families from a number of different crosses. Table 1 shows the correlations between height and disease. Twelve of the 13 correlations were negative and many were individually significant at $P = 0.05$ or less.

In addition to the variation that was correlated with height, there was substantial variation that was independent of height. Some of this was heritable and can be used to breed resistant cultivars of any height, though with increasing difficulty at reduced height (2).

Variation in height and in resistance to S. nodorum were continuous: The effects of individual genes for height and for resistance could not be detected. If there are numerous genes of small effect, pleiotropy is more likely than linkage to be the cause of the observed correlation. Furthermore, several mechanisms for pleiotropic control of height and resistance can be proposed: (1) taller plants are more remote from soil-borne inoculum (this is probably relevant in crops, but not in our inoculated trials); (2) taller plants create a less favorable microclimate for S. nodorum (this is also probably relevant in crops, but not in our small plots); and (3) genetically taller plants have greater vigor, which imparts resistance to S. nodorum (the evidence for this is outlined below).

Among random F_3 families from a given cross, height was always positively correlated with grain yield. This correlation probably resulted from the segregation of minor genes with pleiotropic effects on height and yield. We suggest that they also had a pleiotropic effect on resistance to S. nodorum. Their primary effect, which can be designated vigor, may be manifested in three ways: tall growth, high grain yield, and resistance to a pathogen that is known to thrive on weakened tissue.

By contrast with these minor genes affecting height, the major dwarfing gene Rht-2 was known to have approximately neutral effect on grain yield.

[1]Plant Breeding Institute, Trumpington, Cambridge CB2 2LQ, England.

Table 1.--Relationship between height and incidence of S. nodorum for random F_3 families from crosses between wheat cultivars. Field trials 1976-82.

Year	Parent cultivars		Height[a]		Percentage of S. nodorum[b]		No. F_3 families	r[c]
	T	S	T	S	T	S		
1976	M. Huntsman	M. Fundin	94	65	11	78	20	-0.53**
	M. Ranger	M. Fundin	89	65	52	78	20	-0.50*
	M. Huntsman	M. Ranger	94	89	11	52	20	-0.64**
1977	VPM 1	M. Fundin	111	70	3	46	20	-0.85***
	VPM 1	M. Ranger	111	95	3	16	20	-0.48*
1979	Bonns	M. Ranger	114	98	3	26	20	-0.53**
	Atlas 66	M. Ranger	123	99	16	44	20	-0.53**
1980	M. Huntsman	M. Fundin	104	77	9	24	78	-0.40***
1981	M. Huntsman	M. Fundin	108	77	21	68	118	-0.21*
	M. Huntsman	M. Fundin	109	76	13	78	21	-0.17
	M. Huntsman	M. Ranger	109	105	13	68	21	-0.13
1982	Zorba	M. Fundin	92	63	23	63	21	-0.31
	Zorba	M. Ranger	92	88	23	52	21	+0.36

[a] At maturity (cm). T, taller parent. S, shorter parent. M, Maris.
[b] Percent cover of leaves by S. nodorum lesions at approximately growth stage 75.
[c] Correlation coefficient between percentage of S. nodorum and height for F_3 family means.
* $P<0.05$, ** $P<0.01$, *** $P<0.001$.

The vigor hypothesis would therefore predict a similarly neutral effect on resistance to S. nodorum, in contrast to the usual tendency for taller plants to be more resistant. This was tested for a cross between a tall cultivar and a dwarf possessing Rht-2. Seventy-eight random F_3 families were classified for their Rht-2 genotype by observing the response of seedlings to gibberellic acid. In the field (fig. 1), the 19 families that were homozygous for Rht-2 were on average 13 cm shorter than the 18 families that were homozygous for the alternative allele, Rht-2; the 41 families that segregated for Rht-2 had an intermediate mean height. Despite this large effect of Rht-2 on height, it had no effect or reaction to S. nodorum. Within each group of families of similar Rht-2 genotype, there was substantial variation in height (the result of segregation of the minor genes). This was, as usual, negatively correlated with the incidence of S. nodorum. These are the results predicted by the vigor hypothesis.

A consequence of the relationship observed is that, for plants of a given height, those with Rht-2 tend to be more resistant to S. nodorum than those without. Rht-2 enables breeders to select for shorter straw without incurring the usual penalty of susceptibility to S. nodorum. Selecting the taller Rht-2 plants--"tall dwarfs" (1) allows the minor genes to promote high yield and resistance to S. nodorum, while exploiting the major dwarfing gene to avoid excessively tall straw.

Cultivars with resistance to S. nodorum show some tendency to be resistant to S. tritici also, although there are many exceptions. Again, this correlation is no proof of a genetic relationship between the two resistance characters: for example, selection by breeders for resistance to both diseases might be the cause. Groups of 20 random F_3 families from each of four crosses between contrasting varieties were therefore tested in separate field trials inoculated with each disease. There was a positive correlation between resistance to S. nodorum and resistance to S. tritici for each cross (table 2), indicating some linkage or pleiotropy between the two resistance characters. This was not the result of a common relationship with height, because there was no significant correlation of resistance to S. tritici with height. Unlike S. nodorum, S. tritici does not thrive on weakened tissue, so a correlation between resistance and vigor is not to be expected for this species.

Although correlations were observed between height, yield, resistance to S. nodorum, and resistance to S. tritici, none of them were very strong. There was substantial variation in each character that was independent of the others.

Fig. 1. Relationship between height and incidence of S. nordorum for 78 random F_3 families from the cross between Maris Huntsman and Maris Fundin. Families are classified by Rht-2 genotype (seg. = segregating). Means for each class are indicated by arrows on axes. Ranges of height for each class are indicated by values for extremities of each regression line. Field trial, 1980.

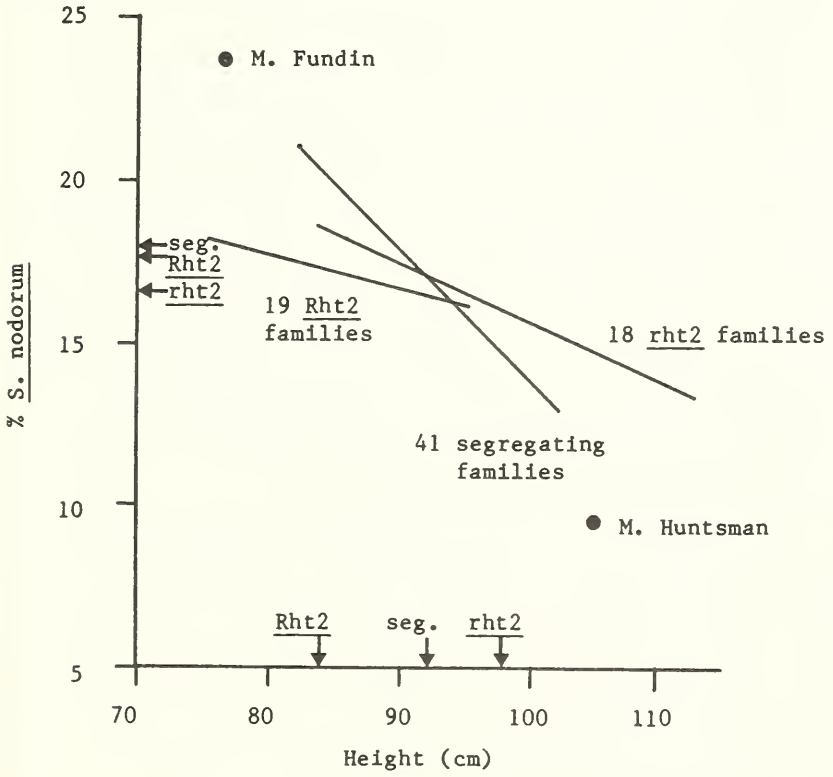

Table 2.--Relationship between incidence of S. nodorum and S. tritici for random F_3 families from crosses between wheat cultivars. Field trials 1981-82.

Year	Parent cultivars T	Parent cultivars S	Percentage of S. nodorum[a] T	Percentage of S. nodorum[a] S	Percentage of S. tritici[a] T	Percentage of S. tritici[a] S	No. F_3 families	r[b]
1981	M. Huntsman	M. Fundin	13	78	29	52	21	+0.49*
	M. Huntsman	M. Ranger	13	68	29	46	21	+0.40
1982	Zorba	M. Fundin	23	63	2	5	21	+0.55**
	Zorba	M. Ranger	23	52	2	7	21	+0.28

[a] Percent cover of leaves by lesions at approximately growth stage 75.
[b] Correlation coefficient beween percentage S. nodorum and percentage S. tritici for F_3 family means.
* $P<0.05$, ** $P<0.01$.
T, taller parent. S, shorter parent. M, maris.

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SCOTT - SPEAKER

Q. Boyd: You mentioned about possibilities of segregating for maturity. I was struck by your specific views of Rht-2. Can you try the same with Rht-1?

A. The maturity effects, we have looked at that, we looked at it in just the same detail as we looked at height, and we tend to find that early maturity and shortness go together, and that early maturity also tends to go with susceptibility. However, the relationship is less close, somewhat less close than with height. But we do find that they tend to go together. So, we think that in part, early maturity is also effected by the Rht genes. So far as Rht-1 or any other Rht gene is concerned, we just haven't looked yet.

Q. I understand from the study of Rht-2 semidwarf genes they do effect the plant.

A. We have not found that yet.

Q. I read an article recently from PBI and ask you to respond. They use the term in there that they were selecting for tall dwarfs. Could you respond to that?

A. That's exactly what I think it is. What is meant by selecting tall dwarfs is, first of all, to fetch the dwarf genotypes, the major dwarfing genotype of Rht-2. But in the essence to the relationship between yield and height, there is a significant positive correlation.

Q. Robin Wilson: I'm concerned about your term pleiotropism when talking of association between nodorum resistance as opposed to linkage.

A. Well, we cannot actually distinguish between the genetic mechanisms and their linkage. We prefer pleiotropism for several reasons. One reason is because we think we're dealing with a number of genes rather than fewer genes with major effects. If you're going to propose linkage, you're going to have to propose that there are a lot of linkages between genes for maximum height and genes for maximum yield or resistance to nodorum. And it just seems less likely to me that there is going to be that much linkage.

Q. Eyal: In the study that we have, which is different slightly from yours, I am happy that on Septoria tritici we agree on the linkage, and I think that we are becoming stronger in this case. Working sources were quite diverse, in which case, taking in both Rht-2 and Rht-1, still there was no justification for linkage. In the case of relationship between disease and height, we got some indication for low association with maturity. Therefore, I think we just cannot exclude this

factor from this analysis. It has to be an integral part because both of them are acting maybe together. I'm not speaking genetically but in the expression of single genes. Between height and disease there was no linkage, but we found a correlation of 0.2 between disease and maturity. There are too many genes operating at the same time, so you couldn't segregate the effect of a single gene and combine them together. Therefore, I think just for convenience, you just put in some indication for linkage and not more than that. Then you have to go through much more precise experiment totally using the different genes and expanding the diversity of the details, other than taking turns you have to test your hypothesis.

A. It is unlikely that there are that many linkages, and hypothesis is circumstantial evidence.

Q. Wilson: I have a two-part question. Firstly, I find it rather intriguing that there should be only one major gene. Could you comment on that? I wonder if the confusion created by height and maturity differences influences interpretation of genetic studies of nodorum and tritici?

A. In relation to the latter point, I think that maturity and height could affect genetic interpretation of resistance. In relation to the Swiss study, we couldn't reproduce the 3 to 1 ratio.

Q. Eyal: If we are conducting a careful genetical study using only a single isolate, I think we are trying to simplify it by putting the blame too much on the lateness in maturity. I think the genetic factors are there, but whether we can distinguish and separate the effect is the problem. One thing that can be done that we have tried recently, is to express the host response in terms of a multiple regression in which you don't need all the effects together. This regression would summarize the combined effects of all the components; severity, maturity, growth stage and maybe the height of the plant. In regard to nodorum, we have been readily able to identify sources of resistance and compare some of their reactions in which the seedling and the mature plant correspond very well. We found moderate interaction between the host and the pathogen, but no specific genetical studies were conducted.

A. I tend to agree with you in relation to Septoria tritici that there seems to be much more indication of linkage involved.

Eyal: We couldn't verify that there was linkage in tritici.

PROGRESS IN BREEDING FOR SEPTORIA TRITICI RESISTANCE IN SEMIDWARF SPRING WHEAT AT CIMMYT[1]

C. E. Mann, S. Rajaram, and R. L. Villareal[2]

INTRODUCTION

Speckled leaf blotch of wheat, caused by Septoria tritici Rob. ex Desm., occurs worldwide (7, 8, 13, 15, 16, 22, 23, 26, 27) and is of economic importance due to the reported possible 30 to 50% yield reduction it causes (8, 12, 14, 17, 24, 25, 33). The increase of the disease incidence was reported to be largely due to the introduction of high-yielding, short-strawed varieties susceptible to leaf blotch. Changes in agronomic practices and the extension of wheat cultivation into humid regions where wheat had not been previously grown commercially have also contributed to the spread of Septoria (19, 20, 24, 25).

Varietal resistance, chemicals, and suitable practices are major control measures for the disease. Crop rotation and other sanitation measures (e.g., burning of infested straw) and the destruction of alternate hosts all contribute to the reduction of primary inoculum in the field.

Breeding for resistance offers the most promising disease control measure. The emphasis of CIMMYT's bread wheat breeding program on disease resistance was expanded in the 1970s to include resistance to S. tritici, and the search for resistance has been very successful. Many lines of spring and winter wheats have shown high resistance in both field and greenhouse tests under natural and artificially induced epidemics. The progress achieved in these breeding efforts is discussed in this paper.

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SOURCES OF RESISTANCE USED BY CIMMYT

Some of the sources of resistance to S. tritici used in the CIMMYT breeding program are presented in table 1. This table includes only those lines that resulted in semidwarf spring wheats with good agronomic type and good yield and/or tolerance to aluminum toxic soils.

Primarily three sources were used successfully for the introduction of resistance: a) Russian winter wheats, b) lines from the southern wheat-growing area of Brazil and from Argentina, and c) to a lesser extent, lines from the United States.

The resistance of these lines was identified and/or confirmed in different countries. Special attention was given to information coming in every year from specialized programs, such as those in Israel and Brazil.

BREEDING METHOD AND TESTING SITES IN MEXICO

Breeding was conducted in Mexico using the pedigree method. Single and top crosses were used. Top crosses were needed to increase the proportion of spring germplasm in winter x spring single crosses, so that progeny would not require vernalization and extended hours of light.

In all segregating generations, single plant selections were made alternately at two sites: Toluca, 2640 m above sea level, 19° north latitude, is conducive to the development of a range of diseases. S. tritici occurs naturally to a limited extent, but artificial inoculation is relatively easy and results in good epidemics every year. The two techniques used for inoculation by our pathologists are: a) distribution of infected straw from the previous cycle between the rows and, b) use of liquid media with spores collected from different parts of the country. The other site, Cd. Obregon, 40 m above sea level, 29° north latitude, is relatively disease free and allows selection for good agronomic type. Advanced lines have also been tested by our pathologists at a third site, Patzcuaro, which has good natural epidemic of Septoria every year.

Table 1.--Source of resistance to Septoria tritici used in CIMMYT's Breeding Program.

	Reference to reports of resistance		Reference to reports of resistance
a) Russian Winter Wheats		LAGOA VERMELHA	31
AURORA	4 32	TZPP	9 28 29 33
KAVKAZ	4 15 32	IAS 62	1 2 31 32
BEZOSTAJA 1	4 32	CNT 7	3 6
		CNT 8	4
b) Spring Wheats from Southern Cone		GABOTO	29
IAS 55	5 30 32		
IAS 58	1 2 30 31 32	c) Spring Wheats from U.S.A.	
PF70254	1 30 31 32	CHRIS	31 32
MARINGA	1 3 31	ERA	31 32
CARAZINHO	2 9 15 29 30	FRONTANA-KENYA 58 x NEWTHATCH	32
IAS 63	4 32		

This multilocation breeding allows selection for a broad range of characters at each stage of the breeding cycle.

ADVANCED LINES DERIVED FROM SEPTORIA RESISTANT SOURCES

Table 2 lists lines that evolved from the breeding process described above. All are semidwarfs and provide acceptable yields. The disease reading was done according to M. Van Ginkel (personal communication), a method CIMMYT staff finds informative and easy for technicians to use. Both digits are taken independently on a scale from 0 to 9. The first digit indicates the height on the plant to which the disease has moved, as was suggested in the method developed by Saari and Prescott (21). The second digit indicates the percentage of area covered by lesions on diseased leaves. Two readings were taken to assess disease progress. All resistant lines showed lower initial readings and considerably slower progress of the disease than the susceptible check.

A second set of materials was developed for areas with aluminum toxic soils (table 3). In most of these areas, S. tritici also occurs. The yield potential of these lines is still relatively low due to undesirable agronomic characters of the Septoria resistant and aluminum tolerant Brazilian parent in these crosses. Nevertheless, these lines proved to be a step forward in improving yield for these areas.

The disease resistance of some of the lines presented in tables 2 and 3 has already been confirmed through reports from cooperators (4, 10, 11). Recent lines (i.e., lines with higher CMnumbers) are now distributed through CIMMYT's international nursery system (International Septoria Observation Nursery, Inter-

Table 2.--Septoria tritici resistant semidwarf bread wheat cultivars with good yield and good agronomic type.

	Toluca, 1982			Cd. Obregon, 1982-83
	Sept. Tr.[1]		Height cm[2]	Yield[3] : Kg/Ha
KVZ-HD2009 SWM2894-1M-1Y-1M-2Y-0M-0MM	22	36	95	6198/6389 CN079
KEA"S" CM21335-C-9Y-3M-1Y-1Y-1Y-0B	27	32	90	5763/6517 URES
KEA"S" CM21335-C-9Y-3M-1Y-1Y-1Y-0B-2KE-0Y	25	33	85	6111/6517 URES
KEA"S" CM21335-C-9Y-3M-1Y-1Y-1Y-0B-6KE-0Y	26	33	90	6194/6517 URES
MAYA"S"-MON"S" CM29251-3M-17Y-1M-1Y-1B-0Y	22	34	95	5719/6554 URES
MAYA"S"-MON"S" CM29251-3M-17Y-4M-0Y-35Y-0B	25	32	90	5720/6554 URES
KVZxBB-CHA/TRM CM30832-Z-3Y-1M-4Y-1M-0Y-1PTZ-0Y	21	22	95	5093/6517 URES
BOW"S" CM33203-H-8M-8Y-1M-1Y-1M-0Y-1PTZ-0Y	25	35	95	6096/6611 URES
BOW"S" CM33203-K-9M-2Y-1M-1Y-1M-0Y	22	34	80	5796/7117 URES
BOW"S" CM33203-K-9M-9Y-4M-4Y-1M-0Y	28	33	90	5565/6517 URES
BOW"S" CM33203-K-10M-7Y-3M-2Y-1M-0Y	12	32	85	6759/7117 URES
SNB"S" CM34630-D-3M-3Y-1M-1Y-0M	16	16	95	5787/7117 URES
SNB"S" CM34630-D-5M-2Y-1M-1Y-0M	23	36	95	5844/6561 URES
SNB"S" CM34630-D-5M-2Y-1M-1Y-1M-1Y-0M	25	34	90	6509/6611 URES
SNB"S" CM34630-D-5M-2Y-1M-1Y-2M-2Y-0M	27	34	90	6083/6611 URES
GOV-AZxMUS"S" CM41257-1-8M-1Y-1M-2Y-2M-0Y-0PTZ-2PTZ-0Y	24	35	90	5113/6389 CN079
BON-YR/F35.70 x KAL-BB CM41860-A-5M-2Y-2M-1Y-0M	25	26	90	5793/6517 URES
CN067-MFD x MON"S" CM43339-C-1Y-1M-2Y-1M-1Y-0B	23	34	100	5491/6389 CN079
LIRA"S" CM43903-H-4Y-1M-1Y-3M-2Y-0B	12	36	80	6650/6474 GLEN

Table 2.--continued.

	Toluca, 1982			Cd. Obregon, 1982-83
	Sept.	Tr.[1]	Height cm[2]	Yield[3] : Kg/Ha
LIRA"S" CM43903-H-4Y-1M-1Y-3M-3Y-0B	11	33	85	6335/6715 GEN
LIRA"S" CM43903-H-4Y-1M-1M-2Y-1M-1Y-0B	12	35	85	6406/6715 GEN
ANA-HUAC"S" CM49258-2Y-2M-3Y-0Y	23	33	90	6683/6715 GEN
MAD"S"-BJY"S" CM49640-3M-1Y-1Y-3M-1Y-1M-0Y	15	25	95	5837/7087 URES
MAD"S"-BJY"S" CM49640-8M-1Y-1Y-1M-2Y-1M-0Y	14	14	95	5752/7087 URES
BJY"S"-PRT"S" CM50323-13M-2Y-2Y-1M-2Y-1M-0Y	11	21	95	5587/7087 URES
MON"S"xKAL-BB CM52368-7M-2Y-2Y-1M-1Y-1M-0Y	12	32	95	6037/6596 URES
DOVE"S"-TSI CM58952-3Y-1M-1Y-2M-0Y	12	33	90	5780/6313 URES
TTR"S"-TTM"S" CM58956-9Y-3M-2Y-5M-0Y	13	33	85	5537/6313 URES
GJO"S"-TRMxBADIA-HUAC"S" CM60767-C-1Y-1M-1Y-1M-0Y	11	32	90	5378/6124 URES
GJO"S"-TRM x BADIA-HUAC"S" CM60767-C-1Y-1M-1Y-2M-0Y	13	32	90	4954/6124 URES
(COQ"S"-F61.70xCNDR"S"/OLN)PHO"S" CM60907-K-1Y-2M-1Y-2M-0Y	12	23	100	4783/6124 URES
BOW"S"-NAC CM61755-10Y-5M-1Y-1M-0Y	26	26	100	5428/6124 URES
GLL-AUST: 161.157xCN067-NO/VEE"S" CM62000-2Y-5M-1Y-1M-0Y	25	34	85	5404/6124 URES
MN72360-SNB"S" CM62067-7Y-4M-1Y-1M-0Y	24	33	90	6256/6748 MYNA"S"
PRL"S"-PAM"S" CM62241-5Y-1M-4Y-2M-0Y	25	32	95	5589/6748 MYNA"S"
TZPP (Resistant check)	21	34	110	
CN079 (Susceptible check)	44	95	80	

[1]Two readings were taken 75 and 96 days after sowing.
Cultivars with a reading of > 37 in the second reading were excluded.
[2]Cultivars with a height of > 105 cm were excluded.
[3]Cultivars yielding more than 1.5 t/ha below the best yielding check of the respective trial were excluded.

national Bread Wheat Screening Nursery, and Aluminum Screening Nursery).

CURRENT BREEDING PLAN

To further broaden the adaptability of CIMMYT lines, an effort is being made to introduce at least a moderate level of *S. tritici* resistance into all CIMMYT bread wheat breeding material, as has been done in the past with the three rusts. Inoculation of all segregating material in 1981 led to a drastic reduction of breeding material for some generations. However, the general level of resistance to *S. tritici* has been improved, and CIMMYT is continuously making specific crosses with highly resistant lines to further improve *S. tritici* resistance.

CONCLUSION

Through a multilocation breeding effort, a considerable number of resistant, semidwarf, good-yielding spring wheat lines are now available to breeders, as well as for release as varieties by national programs.

The hypothesis put forth earlier (18) that the semidwarf character of spring wheats is closely linked to *S. tritici* susceptibility has been clearly refuted.

Table 3.--*Septoria tritici* resistant semidwarf bread wheat cultivars with good agronomic type and tolerance to aluminum toxic soils.

	Toluca, 1982			Cd. Obregon, 1982-83
	Sept. Tr.[1]		Height cm[2]	Yield[3] : Kg/Ha
IAS62-ALDAN"S" CM47049-9M-3Y-2F-2Y-0Y	24	34	100	4252/6781 VEE"S"
PF70354-ALD"S" CM47090-13M-1Y-1F-701Y-9F-704Y-5F-0Y	24	35	95	4304/6782 VEE"S"
PF70354-MUS"S" CM47091-7M-1Y-1F-1Y-0Y	12	33	90	4456/6781 VEE"S"
(IAS58-IAS55xALD"S"/MRNG)ALD"S"-IAS58.103AxALD"S" CM55517-B-1F-701Y-1F-707Y-2F-0Y	23	25	105	4772/7096 URES
PF70534-ALD"S"xMES"S" CM57597-CC-2Y-1Y-4M-0Y	35	37	100	5176/7096 URES
[MRNG(NAD-TORxPCH/BLT"S"-MES"S")]PAT72195(2)-ZP"S"xALD"S"-EMU"S" CM57616-A-3Y-1Y-1M-2Y-5M-0Y	22	34	105	3987/7096 URES
[MRNG(NAD-TORxPCH/BLT"S"-MES"S")]PAT72195(2)-ZP"S"xALD"S"-EMU"S" CM57616-A-3Y-1Y-7M-1Y-1M-0Y	23	35	105	3965/7096 URES
(ALD"S"/FKN-H570.71xFKN)MAD"S"-CNT7 CM58259-A-1Y-1Y-3M-1Y-1M-0Y	24	35	90	4900/7096 URES
BNQ"S"-CNT8xALDAN"S"-IAS58 CM58323-E-1Y-2Y-5M-1Y-5M-0Y	21	33	105	4093/6126 URES
PEG"S"-PF70354(KAL-BBxALD"S"/MRNG) CM58340-A-1Y-3Y-2M-2Y-0M	27	37	100	4181/6126 URES
(CQT-AZxIAS55-ALD"S"/ALD"S"-NAFN)PJN"S"-PELSL 1276.69 CM58478-B-2Y-1Y-2M-1Y-0M	26	36	95	4065/6385 URES
JUPATECO (Susceptible check)	34	67	85	

[1]Two readings were taken 75 and 96 days after sowing.
Cultivars with a reading of > 37 in the second reading were excluded.
[2]Cultivars with a height of > 105 cm were excluded.
[3]Cultivars yielding less than 4.0 t/ha were excluded.

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MANN - SPEAKER

Q. Hosford: What is the current status of the karnal bunt problem?

A. In Mexico, karnal bunt was found in the Sonora area this last year. The only country that so far has taken any action is the United States. I think there have been a number of meetings, 10 or 12, since last March on this topic. We expect now, that experimental material from Mexico will be allowed into the United States under a permit system very similar to that of flag smut with possibly one or two additional requirements. At the moment, the requirements are not finalized. Seed for export from Mexico was handpicked, any karnal bunt infections have been treated with PCNB.

EVALUATION OF BREAD WHEAT GENOTYPES FOR THEIR RESISTANCE TO SEPTORIA TRITICI BLOTCH

Hailu Gebre-Mariam and Getinet Gebeyehu[1]

Septoria tritici blotch (Septoria tritici Rob. ex Desm.), caused by Mycosphaerella graminicola (Fuckel) Schroeter, is one of the major foliar diseases on breadwheat in Ethiopia. The Holetta Research Station is identified as an important screening location for this disease. The experimentally estimated yield loss due to septoria tritici blotch ranges from 6 to 39%.

In the present study, 100 genotypes were evaluated at Holetta and two other locations to assess degree of resistance at different growth stages. Based on this result, four categories of genotypes were identified and, in this report, each class is represented by five varieties (table 1).

THE WEATHER AND SEPTORIA DEVELOPMENT

Very little disease developed during the first 2 months after planting. This period, which extended from June 20 to August 20, showed a continued decrease in maximum and increase in minimum temperatures (fig. 1). The sunshine hours decreased from 4.7 to 2.4, while relative humidity increased from 48 to 84%, and monthly rainfall increased from 84.9 to 284.5 mm (figs. 1 and 2).

Compared with July and August, September showed a substantial increase in disease development. This period, which coincided with growth stages of 6 and 7 for most lines, showed a decrease in the amount of rainfall (284.5 to 124.6 mm), relative humidity (84 to 66%), and minimum temperature (10.3 to 7.4°C), and an increase in maximum temperature (17.8 to 20.9°C) and a significant increase in sunshine hours (2.4 to 7.0 hr) (figs. 1 and 2). Nevertheless, no flag leaf was attacked by the end of September with the exception of a few highly susceptible early maturing lines (e.g., Deres). During October, when most lines were at milk and dough development stages, the septoria tritici blotch development was critical. This period, which was accompanied by high maximum temperature (22°C), low minimum temperature (2°C), a monthly rainfall of 42.2 mm, high sunshine hours (9.7 hr/day), and low relative humidity (42%), resulted in severely infected flag leaves and ears.

TYPES OF RESISTANCE OR TOLERANCE

Varieties were categorized into different types of resistance on the basis of the onset of septoria tritici blotch and its development in relation to crop growth stages.

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Table 1.--Comparative evaluation of four groups of breadwheat varieties for their response to Septoria tritici blotch at different growth states.

Variety	DH	HT	G4 (R)	G5 (R)	G6 (R)	G7 (R)	G8/9 (9)	F%	KW	KQ	PY
				Group 1		Resistant Lines					
HAR 110	68	90	-	0	1	1	4	t	34	2	1.5
HAR 111	68	115	-	-	0	1	6	10	35	2+	1.8
HAR 57	83	110	0	-	2	-	6	10	46	2	2.5
SUB 29	96	110	1	1	2	-	5	t	38	2+	1.6
KUP 705	85	105	0	-	2	-	8	80	37	1	0.3
				Group 2		Tolerant Lines					
HAR 172	85	120	3	3	3	-	9	80	37	2	0.1
HAR 174	76	135	-	2	3	-	8	40	47	1	1.3
ET624D1	80	130	1	1	3	-	4	t	44	2	0.5
WEIBULL	90	150	2	-	3	-	4	0-t	38	1-	0.1
ET12D4	73	85	-	2	3	-	9	80	32	1	2.9
				Group 3		Highly Tolerant Lines					
YAL 564	80	135	2	3	4	-	9	80	44	1-	2.7
TAS 879	80	100	3	4	4	-	8	50	39	1-	2.7
4328	73	125	-	2	3	4	9	70	36	1	2.6
ET620B1	80	105	1	2	4	-	8	40	38	2+	1.3
IAS20	77	140	-	2	3	4	8	70	38	1-	5.7

Table 1.--continued.

Variety	DH	HT	G4 (R)	G5 (R)	G6 (R)	G7 (R)	G8/9 (9)	F%	KW	KQ	PY
				Group 4		Susceptible Lines					
HAR 118	78	95	4	4	5	-	9	90	25	3	3.1
PAN 504	80	100	3	4	4	-	9	90	36	3	2.8
EDC 1138	76	130	-	4	5	-	9	90	27	3+	3.5
OLAF	83	100	3	3	5	-	9	80	24	3-	4.4
DERES	45	110	-	3	4	-	9	90	31	3+	4.7

DH = Days to head

HT = Plant Height (cm)

G4 - G9 = Growth stages 4 to 9 (Feekes' scale)

(R) = Septoria scoring using 0-5 scale

(9) = Septoria scoring using 0-9 scale

F% = Percent of flag leaf infection at maturity

KW = Weight/kernel in mg

KQ = Kernel quality (1-3 scale)

PY = Pycnidia count per square mm of the flag leaf

Resistant Genotypes

Under Holetta conditions, lines with scores of 2 or less, based on the 0-5 scale at milk development stage (G.S. 7), were considered to be resistant to septoria tritici blotch. The resistance of lines in this group was confirmed by the results of kernel weight, kernel quality, low flag leaf infection, and low pycnida count. This group of material is represented by HAR 57, HAR 110, HAR 111, and SUB 29 (table 1).

Tolerant Genotypes

Lines in this group showed a score of 3 by growth stage 7, but had relatively low flag leaf infection, high kernel weight, good kernel quality, and low flag leaf pycnida count per square millimeter. Varieties in this group include ET624D1, Weibull, and HAR 174 (table 1).

Highly Tolerant Genotypes

These were genotypes with scores of 4 or more by growth stage 7, but showed intermediate levels of flag leaf/ear infection, high kernel weight, good kernel plumpness, and relatively low pycnida count. Considering the high level of susceptible reaction of these lines at growth stage 6 or before and observing the high kernel quality attained at the end, it is hypothesized that these genotypes are highly tolerant to the pathogen. We are further investigating the level of infection in relation to yield reduction to substantiate this observation. Some genotypes of interest in this group are IAS 20, TAS 879, ET620B1, and 4328 (table 1).

Susceptible Lines

Under Holetta conditions, susceptible varieties showed a high score of 4 by booting stage followed by a very high level of flag leaf/ear infection, resulting in highly shrivelled kernels (table 1).

Figure 1.--Maximum and minimum temperatures and sunshine hours (ssh) June-Nov. 1982. (hrs).

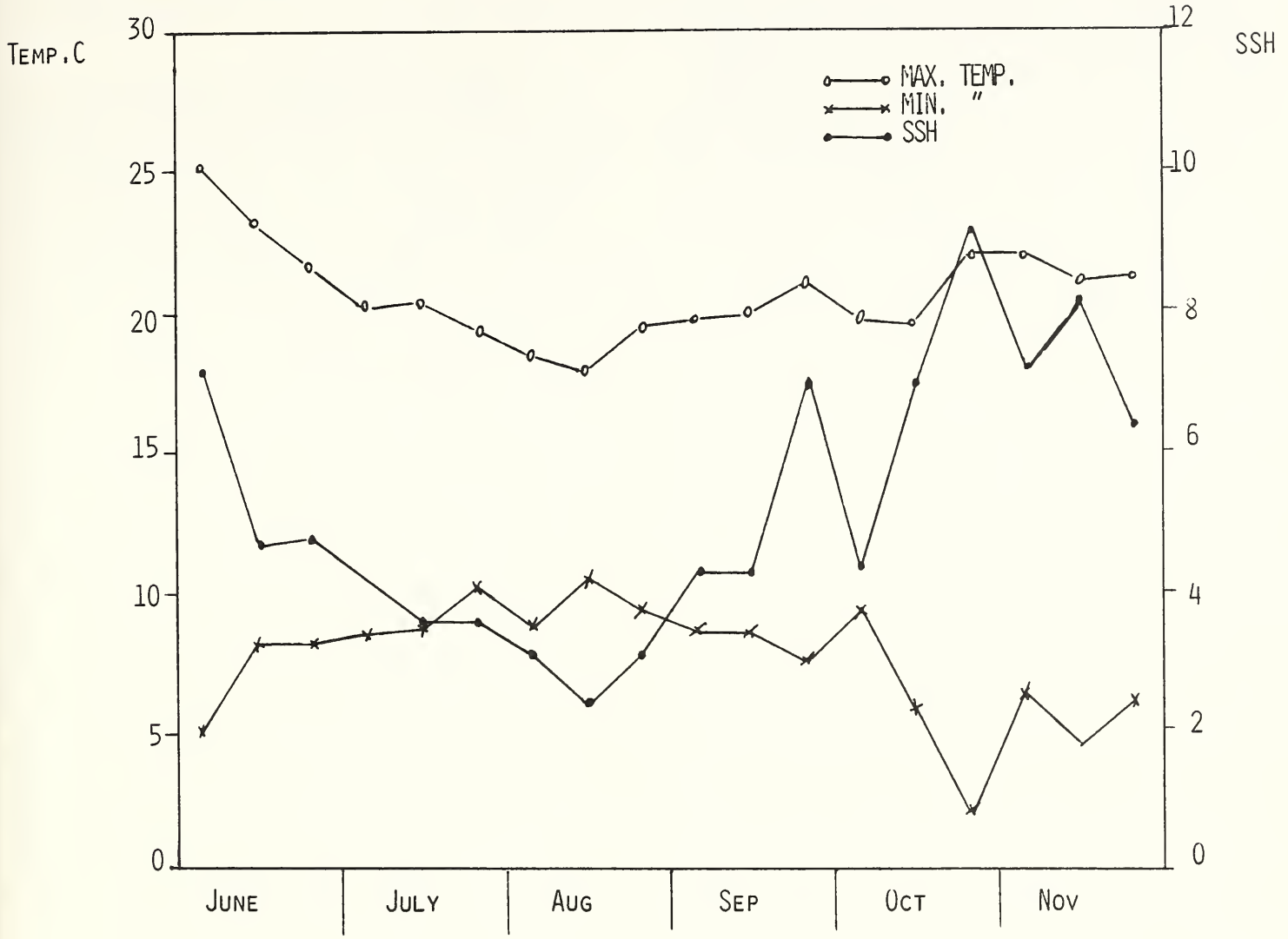
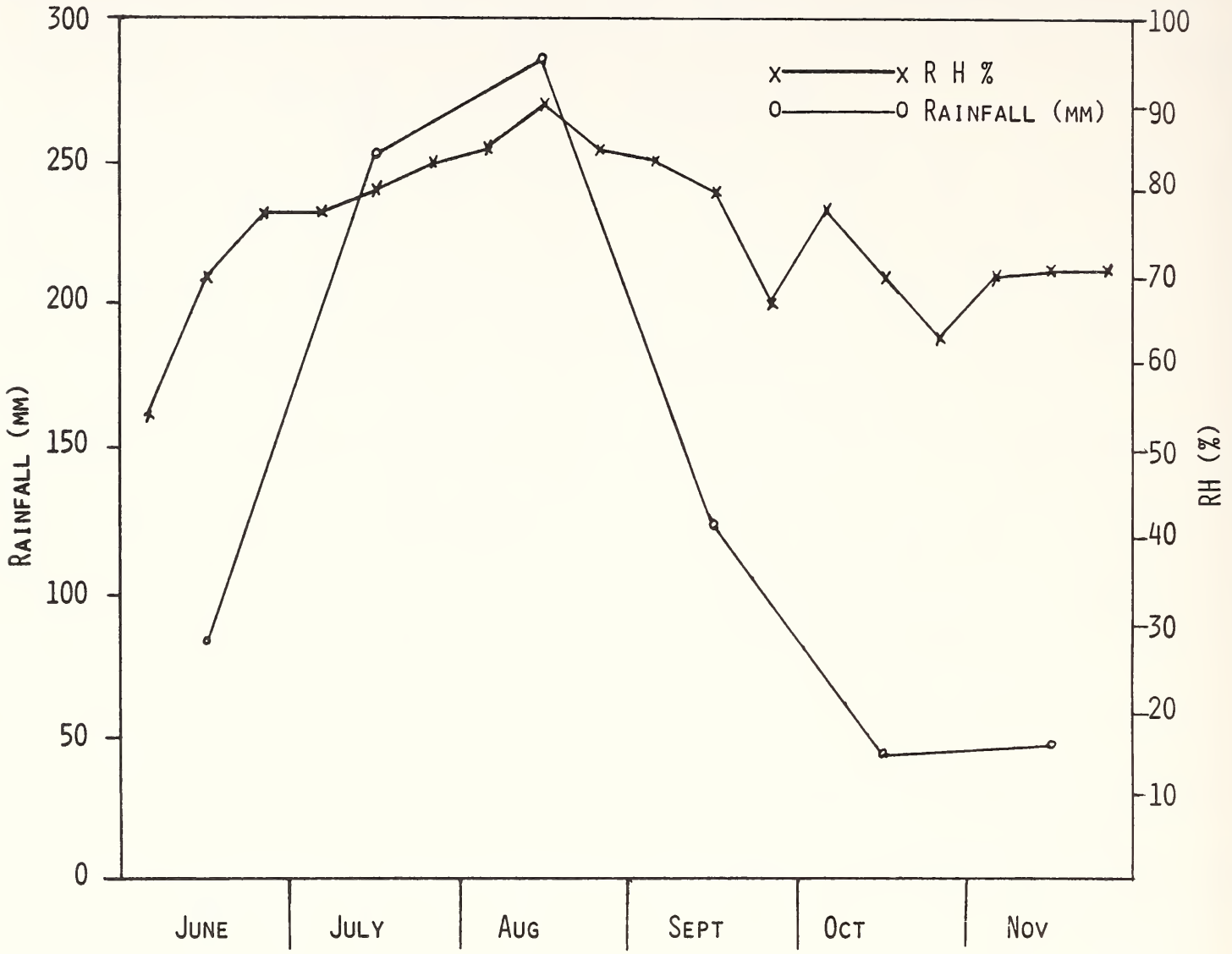


Figure 2.--Rainfall and relative humidity (rh) June-Nov. 1982. (hrs).



RESISTANCE TO SPECKLED LEAF BLOTCH OF WHEAT IN SOUTHERN NEW SOUTH WALES

Barbara Ballantyne[1]

The destructiveness of speckled leaf blotch (SLB) (Mycosphaerella graminicola (Fckl.) Schroeter) in southern New South Wales was recognized sporadically from the 1930's onwards. Sources of resistance were located, but the lack of a reliable screening test inhibited efforts to breed for resistance. The interest of R. H. Martin which began in the 1950's was more sustained. In 1966, he made crosses with the cvs. 'Fleche d'Or' (bread wheat, Triticum aestivum L. em. Thell.), and 'Farro Lunga' (durum wheat, T. turgidum L. em. Thell. var. durum Desf.). He sowed the progeny in early nurseries, which he watered to encourage the disease, and in this way transferred the high level of resistance in these cultivars to a range of lines, some of which are in advanced trials. His earlier material M1696 & M2053) has also been used by other breeders. The cv. 'Teal', which he released in 1972, has partial resistance, the origin of which is not clear (5). Other workers have since joined the Wagga group. J. Kuiper found increases of up to 150% in fungicide-protected plots of the very susceptible cvs. 'Heron', 'Robin', and 'Summit', grown during the early 1970's when conditions were very favorable for SLB (3). He transferred resistance from the durum cvs. 'Lobeiro' and 'Pelissier' to bread wheat (SK lines), which have been further crossed with material resistant to stem rust (Puccinia graminis f. sp. tritici Eriks. and E. Henn.) (J. Kuiper, personal communication). A survey by G. Murray of the wheat Septoria diseases in the State showed that M. graminicola was the predominant pathogen, but Leptosphaeria nodorum Muller and L. avenaria Weber f. sp. triticea T. Johnson were sometimes present (6, 7). He also studied SLB development in relation to different seasonal conditions. May (4) transferred high levels of resistance from triticale (X Triticosecale) to bread wheat and is screening the chromosomes with molecular probes. J. Fisher and A. Khan have used resistant parents in their breeding programs; however, until recently, progress has been hampered by the lack of continued field epidemics. There was a need for a screening test plus information on the genetics of resistance in the host and physiologic specialization in the pathogen (5). This has been my field for the past 4 years.

Laboratory and glasshouse procedures have been developed to enable large scale screening (1). Fresh cultures are begun from refrigerated soil tubes every 4 weeks, and the plates are incubated and stored at low temperatures to minimize subculturing. A vigorously growing culture is used as inoculum for broth (yeast extract:malt extract:sucrose) in Erlenmeyer flasks, which are shaken at 200-250 orbits per minute for 2-2 1/2 days at 18°-19°C. Gelatin (0.5%) is added as a sticker to the pycnidiospore suspension sprayed on the leaves. Plants are kept moist for 4 days then returned to the glasshouse bench at about 19° (day) and 15° (night). The test may be used in a number of ways (1). For example, last season, about 1,500 advanced breeding lines were screened in duplicate. Seedlings at about the three-leaf stage were tested in 10-cm pots. Observations were made on two occasions (2 1/2 and 3 1/2 weeks after inoculation) as some genotypes with partial resistance, for example, Teal, develop the disease more slowly than susceptible lines. When field and glasshouse results were compared, there was satisfactory agreement in one series of lines. However, in another group, there were suggestions that the culture (79.2.1a) used routinely to date, was not virulent on all pedigree groups. Of the lines derived from Farro Lunga and Fleche d' Or, some were completely free of disease in the glasshouse and others were partially resistant.

Glasshouse testing has been of particular benefit in the backcrossing project conducted jointly with Fisher and Martin. Five locally adapted wheats and five sources of resistance have been used, M1696, M2053, Canrock I, IRN 643, and IBO-5-377. In 1981, the first season in which a large scale planting was assessed in a field epidemic, there was a marked contrast between the level of resistance in the backcrossing block and other early generation material. The population derived from the intensive testing and crossing of SLB resistant material contained many lines with good resistance and desirable agronomic type. Other adjacent, previously unscreened populations included a large proportion of susceptible or very susceptible lines. The most advanced material is at BC1 and 2 F_5.

Field screening has been carried out with early sowing, spreader rows, and irrigation. During the 1982 drought, there was no significant disease, but useful levels of SLB generally occur red in the nurseries. Among more than 1,000 introductions screened in recent years, a wide range of wheats have shown at least moderate resistance. To assist interpretation, the results have been listed firstly according to disease score (Rosielle scale) and secondly by maturity as the late and very late-maturing entries may have escaped some disease. Most of the currently grown and recently released Australian cultivars are much less susceptible than those used a decade ago. Of particular note is the cv. 'Banks' which was selected in Queensland in the absence of SLB but has moderate resistance in most outbreaks. The source of its resistance is not clear, although we have known for years that some lines derived from WW15 (syn. 'Anza', 'Karamu'), used widely as a parent, have some limited degree of resistance, for example, 'Oxley' and to a lesser extent, 'Egret'.

Breeders have been supplied with resistant material for their own crossing nurseries. Resistance to at least one of the cereal rusts was present in most of these candidate parents comprising selections from the backcrossing program and introductions, for example, 'Kenya Nyolka', a Bobwhite selection (12 IBWSN 232), and 'Cleo'/'Inia' 66 and 'Tadorna'/'Inia' 66 lines from the University of California, Davis. Most are shorter and/or earlier maturing than sources of resistance identified years ago,

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for example, 'Carazinho', 'Maringa' and 'Romany'. These lines have originated from the Australian Septoria Nursery (AUSEN), coordinated by R. Wilson, Roseworthy Agricultural College, the ECN (Elite Crossing Nursery, coordinated by D. The, University of Sydney Plant Breeding Institute), and lines requested from the Australian Wheat Collection on the basis of overseas reports.

Inheritance of resistance is being investigated. F_1, F_2, and F_3 seed of a wide range of crosses with resistant wheats used locally have been generated, using single typical plants of each parental genotype. Field disease reactions of F_3 lines in 1981 did not suggest simple inheritance, but the varying heights and maturities of the plants and the possibility of different pathogen genotypes may have obscured the situation. The number of plants in the different disease reaction classes were summed for all families in each cross. Crosses with the susceptible parent CSP 44 gave a higher proportion of resistant plants than those with cv. Summit, a more severely affected susceptible tester. The breeding line M1696 gave the greatest proportion of resistant progeny, M2053 showed slightly fewer, and IRN 643 gave the lowest recovery of resistant plants. Glasshouse tests with particular cultures will be carried out. Single seed descent populations have been generated to determine how the partial resistance in cultivars such as Teal and 'Kondut' is inherited, and if it can be improved. It would appear that this resistance of Teal has been stable during the past decade.

The type and extent of variation in the pathogen are being studied. A collection of isolates from many bread and durum wheats from several areas of the State has been stored (mostly in soil, under refrigeration). Glasshouse experiments with cultures derived from single spores have shown variation as reported by other workers (2, 8, 9). The cv. 'Veranopolis', used in some programs as a source of resistance, developed necrosis and pycnidia when seedlings were inoculated with culture 81.85.1d1, but only tip yellowing with others.

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BALLANTYNE - SPEAKER

Q. Scott: You said that some of your highest sources of resistance were less effective in the severe epidemic a couple of years ago. Are you suggesting that that was just because the epidemic was severe, or are you in addition suggesting that the isolates around in that year showed physiologic specialization towards the varities in question?

A. I don't know, there are many possibilities. Before that year, I had collected some wheats all of average sources of resistance except some that were a bit too early. I haven't been able to isolate the problem as well as I thought before. I don't know what happened.

Q. Scott: Do you know that they had additional virulence or were they more pathogenic when you tested them in a less extreme environment for example.

A. I have compared a range of cultures from many localities.

Q. Scott: And are some more pathogenic than others?

A. Oh, definitely.

Q. Scott: So they have increased pathogenicity, but you don't know whether it is host specific pathogenicity?

A. Oh, it's quite specific.

INHERITANCE OF RESISTANCE TO SEPTORIA TRITICI IN WHEAT

R. E. Wilson[1]

Although incorporation of resistance to Septoria tritici Rob. ex Desm. (imperfect stage of Mycosphaerella graminicola (Fuckel) Schroeter) is an objective in many breeding programs, we know little about the number and nature of the genes controlling resistance and how they relate to each other. Simple inheritance has been previously demonstrated in some cultivars. Single dominant genes for resistance have been shown to be present in Lerma 50, P14 (2), Bulgaria 88 (3), Veranopolis (5, 6), Israel 493 = AUS16144 (6), and IASSUL = IAS20 (5). Other modes of inheritance have also been found--two partially dominant genes with additive effect in Nabob (2), a single recessive gene in an unnamed variety (1), and at least three recessive genes in Seabreeze (5). In the only study of the interrelationships of the genes for resistance, it was shown that those in Veranopolis and Israel 493 are independent and the one in Veranopolis is independent of the gene in Bulgaria 88.

It would be useful if linkages of gene(s) for resistance to S. tritici with morphological characters could be detected. The genes present in Veranopolis and Israel 493 were shown to be each independent of the genes governing grain color and awnedness (6).

This paper reports the inheritance of resistance in 28 sources of resistance used in the Roseworthy breeding program and how these relate to the "testers", Veranopolis and Israel 493.

METHOD

Each resistant parent was crossed and backcrossed once to the susceptible crossbred RAC311H and also to the testers. F2 and BC1F1 plants were rated for reaction to S. tritici, using the Rosielle scale (4), and heights were measured. In the next season, F3 and BC1F2 hillplots were rated for S. tritici reaction, and both height and maturity were recorded since it is known that they influence the disease rating. A contingency table analysis was used to see if the interaction of maturity and height with Septoria reaction was significant. If it was, the data were corrected so that each height and maturity level was equally represented in the total. The parents were always grown next to each cross, and the range of reaction to S. tritici on them was used to determine the cutoff point in the analysis.

Where the parents differed for characters such as grain color, awnedness, chaff color, and ease of threshing, these were recorded in that cross.

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RESULTS AND DISCUSSIONS

The results for reaction to S. tritici for the 28 parents and the probable mode of inheritance are shown in table 1. Unfortunately, not all four crosses are present for each parent as mice took their percentage at a number of stages.

There is a predominance of the single dominant gene mode of inheritance although other modes are represented including two that have not been previously found--duplicate dominant genes and a single incomplete dominant. The genes in Bulgaria 88 and Israel 493 are different. When this result is combined with previous work (6), it can be seen that there are at least three different genes. It is proposed that they be designated Slb1 for Bulgaria 88 (with its derivatives Oasis and Sullivan), Slb2 for Veranopolis (and Nova Prata) and Slb3 for Israel 493. The results here are consistant with the findings of Rosielle and Brown (5) for IAS20 and Seabreeze although there is a better fit to a two recessive gene model with this data. However, the gene in IAS20 appears to be different than the one in Veranopolis despite the fact that these cultivars are related and have similar inheritance patterns.

The only linkage to morphological characters detected was the linkage of the resistance genes in Canrock I, IRN643, and (Romany-Gb-Gamenya) with brown chaff. The recombination percentage in each case is approximately 38.5%, suggesting a common gene for S. tritici resistance.

A number of these resistances have been backcrossed further to RAC311H and many provide suitable components for a multiline as well as enabling more exact studies of the genes where the background is similar.

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Table 1.--Totals and probabilities of the models proposed for crosses of resistant parents by susceptibles and testers.

Resistant Parents	RAC311H				2*RAC311H				VERANOPOLIS				ISRAEL 493				Model θ
	F2		F3		BC1F1		BC1F2		F2		F3		F2		F3		
	Σ	P>	Σ	P>	Σ	P>	Σ	P>	Σ	P>	Σ	P>	Σ	P>	Σ	P>	
Aniversario			107	.20	7	✓	48	.20	200	.30	96	.80					SD
AUS22=Aust. hybrid									70	.80	160	.30					SD
Bulgaria 88			84	.05[∠]	21	.50	19	.80					203		82	.30	SD
Canrock I	489	.30	210	.20	72	.80	67	.50	180	.50	146	.10			113	.20	SD
Cotipora			133	.05	19	.30	18	.50									SD
C3228/65 (Brazil)			78	.20	9	✓	9	✓	202	.001	78	.30					SD
[DRP(FN-K58 x N10B/ GB55) NAI60]	376	.10	192	.10	36	.30	31	.80			157	.10			146	.001[β]	SD?
(Farrolunga/Heron)	671	.05	379	.95	50	.30	10	✓	227	.95	105	.80					SD
Gala	555[δ]	.70			53	.80	46	.10									IncD
IASSUL = IAS20	21	.20			39	.50	37	.30	352	.02	290	.50					SD
IBO5-377	339	.90	295	.001	37	.20									88	.05	SDor
		.80		.001		.02										.50	TCD
IRN641=IRN62-641?	666[δ]	.30	89	.20													SD
IRN643=IRN62-643?	364	.70	159	.70	66	.50	63	.30	171	.80	87	.30	207		118	.05	SD
K4500-4 (Kenya)	220	.30	333[δ]	.30	31	λ	24	.50	67	.50	109	.30	23		114	.30	SRec
(LEE-RL2564 x Fr/ IAS54)-6th Isep-ton-105)	331	.70	236	.02	59	.50	53	.30	255	.80	108	.80	233		80	.30	SD
Leone	433	.80	28	.001	56	.10	26				80				77	.001	TD1
		.10		.001		.70		.50				.50				.01	SD2
Malta Yellow	27	.80	26	.001	21	.30	12	.30					68				TD
Nova Prata			126	.20	12	.20			108	Δ							SD
Pavon "S"			59	.30	58	.10	50	.80			118	.50					SD
PF70216			83	.001[β]	12	.50	13	.50	195	.05	127	.20					TD
PF70354			94	.20													SD
Romany			88	.02	39	.80	24	✓									SD
(Romany-Gb-Gamenya)	232	.02[β]	229	.02[β]	46	.30	46	.10	171	.50	119	.50					SD
Seabreeze	520[δ]	.50			45	λ	45	.10									TRec
Temu 113.70	89	.30	54	.30													SD
Tevere			173	.10	40	.50	38	✓			47	.70					SD
Tosca	294	.05	223	.10	69	.90	61	.05							146	.02	SD
Touko Jokioninen	1106[δ]	.50	211	.001	67	.10	28	.30	153	.30	144	.05			112	.001[β]	TCD

θ Models:
SD = Single dominant gene
TD = Two dominants
SRec = Single recessive gene
TCD = Two complementary dominant
IncD = Single incomplete dominant
TD1 = Two dominants at first reading
TRec = Two recessive genes

∠ = Corrected for maturity and height influences
δ = Includes crosses to other susceptibles
Δ = All resistant
β = Excess of susceptibles
λ = All susceptibles

WILSON - SPEAKER

Q. Scott: Could you just go over again how you go about catagorizing your plants into resistant and susceptible in crosses where you don't have an obvious discontinuity in frequency distribution?

A. I'm using a range of reactions in the two parents, and that's how I determine the cutoff point.

Q. Scott: And you were able to confirm the performance by testing?

A. Yes.

Comment by Scott: Based on the parental ranges, your results certainly show heritability of resistance. Nevertheless, it seems to be slightly awkward to argue very precisely about individual genes unless you can detect discontinuities in the frequency distribution.

A GENETIC ANALYSIS OF *TRITICUM AESTIVUM* 'Vilmorin' RESISTANCE TO SEPTORIA LEAF BLOTCH AND PYRENOPHORA TAN SPOT

F. J. Gough and E. L. Smith

A winter wheat cultivar (*Triticum aestivum* L. 'Vilmorin') having resistance to Pyrenophora tan spot and Septoria leaf blotch was crossed with a susceptible one ('Chisholm'). First and second leaves of approximately 20 seedlings in each of 70 F_3 families, grown in 5-cm pots, were spray inoculated with chopped mycelium of *Pyrenophora tritici-repentis* (culture PYOK-14). After inoculation, the plants were kept moist under opaque polyethylene film for 48 hr in a room maintained at about 20°C and with a 12-hr light (75 $\mu E/m^2/sec$) and 12-hr dark cycle. The families were then placed in a greenhouse and scored for reaction to *P. tritici-repentis* 5 days later.

The plants were next vernalized in a cold frame for 40 days where the temperature fluctuated from an occasional high near 30°C during the day to near 0°C at night. After vernalization, plants within each family were transplanted en masse to 12.5-cm pots and placed on a greenhouse bench. At jointing (growth stage 7 Feeke's scale), the plants were inoculated with a conidial suspension ($8x10^6$ conidia/ml) of *Septoria tritici* (culture 83A), covered with a polyethylene and muslin chamber, and kept moist for 96 hr with two clock-controlled humidifiers.

Resistance to tan spot was expressed as discreet dark-brown oval circles approximately 2 mm long and 1 mm wide surrounding tan centers. Susceptibility was expressed as coalescing, irregularly shaped chlorotic and necrotic lesions usually having small brown spots at the points of infection. Leaves of resistant plants exhibited little or no damage outside the lesion areas and were 90 to 95% green. Leaves of susceptible plants exhibited necrosis extending through 90 to 95% of the tissue. Based on the presence of resistant and susceptible plants, the 70 families were scored 16 homozygous for resistance, 37 segregating for resistance and susceptibility, and 17 homozygous for susceptibility. These numbers were a good fit to a 1:2:1 ratio (P = 0.8-0.9) expected for a single gene segregation.

Resistance to Septoria leaf blotch was expressed as relatively limited necrotic lesion tissue and by few pycnidia in the lesions. Susceptibility was expressed as nearly total necrosis of infected leaves and by numerous pycnidia in the necrotic tissue. As with tan spot infection, the 70 families segregated into three groups, as follows: 17 homozygous for resistance, 39 segregating for resistance and susceptibility, and 14 homozygous for susceptibility. These numbers were a good fit (P = 0.5-0.7) to expectations for alleles segregating at a single locus.

An x^2 test for independence indicated (P = 0.02-0.05) that the gene which conditioned resistance to tan spot and the one which conditioned resistance to Septoria leaf blotch may be associated.

GOUGH - SPEAKER

Q. Scharen: Did you tell us where Vilmorin came from?

A. No,I did not. Vilmorin is a French wheat, and should have an identification number. I received it from someone at Texas A&M. I believe there is a woman in France who possibly has been using this particular variety, and she has some of the monosomic series.

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IDENTIFICATION, DISTRIBUTION AND NOMENCLATURE OF THE SEPTORIA SPECIES THAT ATTACK CEREALS

A. L. Scharen[1] and F. R. Sanderson[2]

Septoria is the name commonly applied to over 1,000 form-species of fungi, most of which are plant parasites. Approximately 100 species are parasitic on cereals and grasses. Many are economically important on crops other than cereals.

The two pathogens of the Septoria group that have had the greatest impact on overall agriculture are L. nodorum and M. graminicola, both wheat pathogens. Losses of grain yield in epidemic situations are upwards of 50%, but on a sustained world-wide basis, these two pathogens reduce the total crop by about 2%. In 1982, that loss was estimated to be 9 million metric tons with a value of over $1 billion.

Within the Fungi Imperfecti, Septoria is classified among the Sphaeropsidales. Conidia, termed "pycnidiospores," are produced in variously shaped semiclosed bodies known as pycnidia. When exposed to moisture, the spores often exude from ostioles in wormlike masses, or cirrhi. The teliomorph or sexual stages of these fungi, where known, are associated with the ascomycetes (table 1).

A continuing discussion of the proper taxonomic assignments for those species that overlap in their descriptions has been in progress for more than 100 years (4, 7 and table 2). Important to the study of cereal diseases are the names of at least two species. Recently, J. Bissett (1, 2) has published in Fungi Canadenses two revisions which would place Septoria avenae and S. nodorum in the genus Stagonospora. These have overlapping characters, and some justification can be found for placing them in either genus. In his book, "Diseases of Cereals and Grasses in North America" (7), Roderick Sprague discussed these questions in detail more than 30 years ago. He concluded that researchers and farmers around the world are used to the term "septoria" and its association with certain diseases of cereals. Since there was no compelling reason to mandate a change in 1950, he preferred to leave species that had been in Septoria without change. Participants in the International Workshop on the Septoria Diseases of Cereals, held at Montana State University, August 1-4, 1983, agreed that the taxonomic names of the fungi involved in the Septoria disease complex would be based on their teliomorph state, namely Leptosphaeria nodorum E. Müller, L. avenaria Weber f. sp. triticea T. Johns., and Mycosphaerella graminicola (Fuckel) Schroeter, and that the common names of the diseases be septoria nodorum blotch, septoria avenae blotch, and septoria tritici blotch.

The taxonomy of those Septoria spp. important on cereals places them in the Loculoascomycetes, having fruiting bodies called pseudothecia (table 1; figs. 1 and 3). However, the most common spore-containing structure familiar to students of cereal diseases is the pycnidium of the anamorph form of the organisms (figs. 2 and 4).

Confirmation of the Leptosphaeria group and differentiation from the Mycosphaerella group is always possible. But even after careful examination, some doubt may remain as to which of the Leptosphaerias are present.

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Table 1.--Classification and nomenclature of Septorias that attack small-grain cereals.

EUMYCOPHYTA (True Fungi)		
Class: Ascomycetae (Ascomycetes)		
Subclass: Loculoascomycetes		
(fruiting body a pseudothecium)		
Order - Pleosporales		Dothideales
Family - Pleosporaceae		Dothideaceae
Genus - Leptosphaeria		Mycosphaerella
L. nodorum (S. nodorum)		M. graminicola (S. tritici)
L. avenaria (S. avenae)		
L. avenaria f. sp. triticea (S. avenae f. sp. triticea)		
L. avenaria f. sp. triticea (S. passerinii)		

Table 2.--Descriptive comparisons of pathogens.

Perfect Imperfect	Pseudothecia Pycnidia (u)	Ascospores Pycnidiospores (u)	Septa	Description of lesion
Leptosphaeria nodorum	120-200	23-32x4-6	3	Discolored to brown; often lens-shaped with chlorotic yellow border. Central portion with pycnidia/ pseudothecia.
Septoria nodorum	160-210	15-32x2-4	0-3	
Leptosphaeria avenaria	100-220	19-25x4-6	3	Straw colored to buff. Central portion with pycnidia/pseudothecia.
Septoria avenae f. sp. *triticea*	90-140	26-42x3-4	3-4	
Septoria passerinii	90-150	23-46x2-3	1-3	Indefinite, yellowed, pycnidia dark brown, prominent and numerous.
Mycosphaerella graminicola	70-100	10-15x2-3	1	Straw colored, irregular to rectangular, elongated between parallel veins, often speckled due to prominent numerous pycindia/pseudothecia.
Septoria tritici	60-200	35-98x1-3	3-5	

Figure 1.-- Pseudothecia, asci, ascospores of *M. graminicola*.

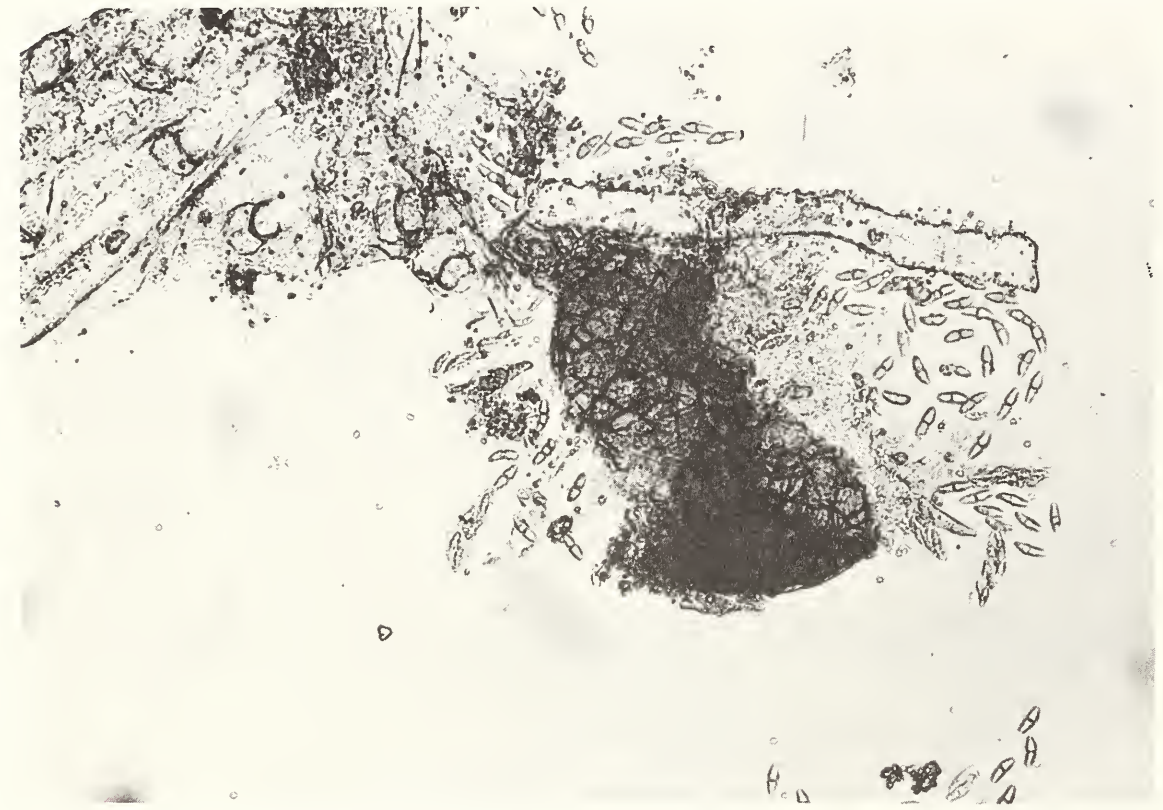

Figure 2.--Pycnidia, pycnidiospores of Septoria tritici.

Figure 3.-- Pseudothecia, asci, ascospores of L. nodorum.

Figure 4.--Pycnidia, pycnidiospores of Septoria nodorum.

On grasses, the host-range of Septoria species tends to be limited by related groups of grass hosts. The different specific groups must have evolved through their associations with certain hosts, with more physiological variation than morphological variation. For example, isolates from wheat, barley, and several forage grasses are nearly identical in morphology (fig. 5) but exhibit strong physiological affinity for one or another host (3, 5, 6). The closeness of grains and grasses to the moist soil and their tendency to grow in dense stands favoring high humidity result in a microclimate favorable to development of many kinds of Septoria on these hosts.

The common names of the diseases are septoria nodorum blotch and septoria tritici blotch. Both pathogens involved in these diseases can parasitize and cause damage on all plant parts from seedling leaves to heads. Disease development can be arrested at any time by the advent of warm, dry weather, but development often resumes if conditions again favor disease.

Symptoms vary according to cultivar, cultural practice, and geographic locale. Under Mediterranean conditions, where spring wheats are grown during the coolest months of the year, M. graminicola is most important. Usually, many pycnidia are produced, making identification simple. In the southeastern part of the United States, and northern Europe, L. nodorum is most common, usually producing an abundance of pycnidia allowing identification with ease; however, in many other wheat-growing areas, both L. nodorum and M. graminicola occur. In the United Kingdom, northern United States, Brazil, and Uruguay, Western Australia and other areas, septoria nodorum blotch and septoria tritici blotch are found together, with fruiting structures of both organisms on the same leaf. In most of these areas, L. avenaria f. sp. triticea occurs as well, sometimes complicating identification. Other fungi that form similar fruiting structures, spores and symptoms are usually present to complicate identification (5 and fig. 5). Thus, field identification without laboratory confirmation is often difficult if not impossible; however, with the preparation of a few slides and examination at about 500x, identities of pathogens can usually be confirmed.

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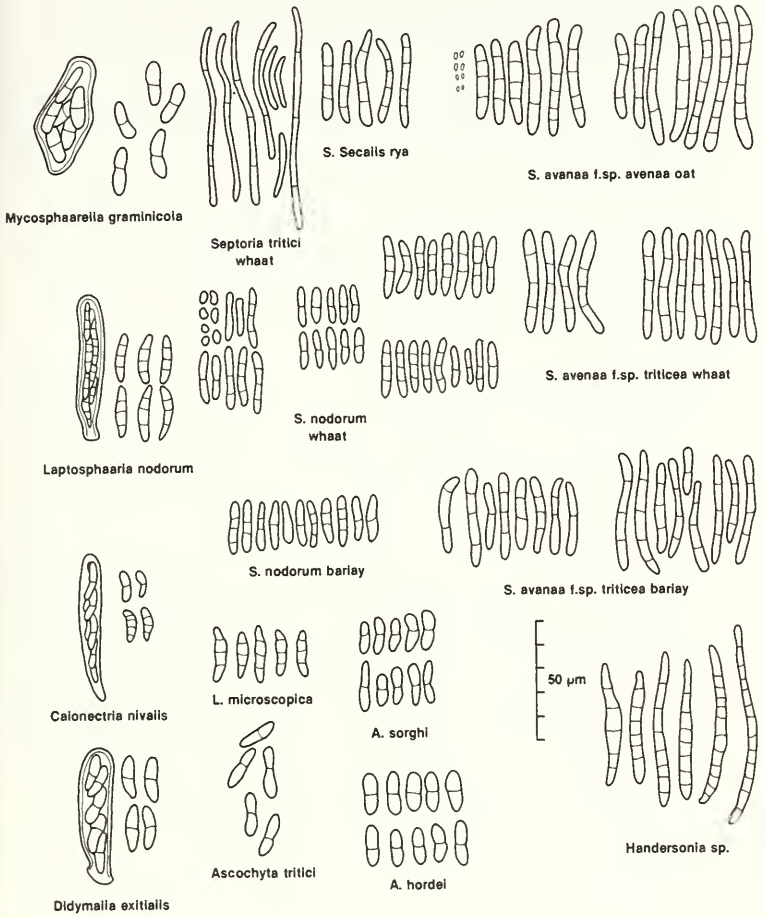
Figure 5.--Septoria-related spore forms on cereals, and other spores that could cause confusion in identification.



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SCHAREN - SPEAKER

Q. Peter Scott: Is there any possibility of using the name S. nodorum, S. tritici, as the common name? In England, any farmer who is aware of the difference between the two species, and there are many farmers who are, have used those terms instead of speckled leaf blotch and glume blotch for Septoria nodorum and Septoria tritici. Is there any problem in using these as the common names?

If you were pressed to give common names to the three septoria species of wheat, what common names would you use?

A. We have used speckled leaf blotch for the disease caused by S. tritici. Since it is the most well-known name, we use glume blotch for the disease caused by S. nodorum. We haven't discussed in enough detail those caused by any of the other septorias to really have decided on a common name.

Q. Barry Jacobsen: As we are getting to the more selective chemicals now, we are going to have to recognize them as nodorum or as tritici. Farmers are going to have to know those terms.

A. It would be very helpful if we could tell all the farmers the same story and if we could get as many of them as possible to use the same names. When you are advising chemical control, you must know which organism is present.

SEPTORIA LEAF BLOTCH OF WHEAT IN CHILE

I. Ramirez and M. Caglevic[1]

Wheat is the most important annual crop in Chile. It is the main source of carbohydrates and protein for the human diet compared with other agricultural foodstuffs.

Among the diseases that affect wheat in Chile, Septoria tritici Rob. ex Desm. has become increasingly important during the last 10 to 15 years in those areas where wheat is cultivated under rainfed conditions within a range of 600 to 1500 mm, or more, precipitation and cool temperatures. Conditions in southern Chile and the central coastal humid region are ideal for natural Septoria epidemics. Septoria tritici was found and described in Chile in 1926. Today S. tritici is considered a limiting factor for wheat production in the areas described above. High disease levels are commonly found causing losses of 7 to 12% in years with minor occurrence and up to 40 to 75% when favorable weather conditions allow severe epidemics to develop.

Septoria glume blotch, Septoria nodorum, also occurs in Chile but is less important than S. tritici. It was first described in Chile in 1966.

Current work includes: a) screening for disease resistance using a wide array of germplasm; b) assessment of losses caused by pathogens; and c) chemical control trials to test types, doses, and time of application of fungicides.

[1]Head, Wheat Project, INIA, Santiago, Chile; and plant pathologist, Wheat Project, INIA, Chile.

Several nurseries at four experiment stations are evaluated each season for Septoria reaction. The material evaluated has been developed by the National Institute of Agricultural Research (INIA) Wheat Project, or are introductions from cooperative nurseries such as International Center for Improvement of Corn and Wheat (CIMMYT), International Septoria Nursery (ISEPTON), Cooperative Septoria Nursery from the Southern Cone Program, and the Field Evaluation Nursery from Bozeman.

During 1981, a moderately severe attack of Septoria tritici was observed, while in 1982, due to an exceptionally rainy season, a most severe epidemic was present in the coastal region and southern Chile. A very effective screening could be done under such conditions. From the nurseries tested at the Hidango Experiment Station, sample listing of entries which showed promisingly low readings is presented (table 1).

Table 2 summarizes notes taken on the 12th ISEPTON at Hidango and the South Central Experiment Station Quilamapu showed lines with resistant reactions at both sites in 1982, with early spring type checks being the most susceptible entries. All screening has been done under natural infection conditions.

Resistance or "tolerance" (escape mechanism) seems related to growth habit and development pattern (spring vs. facultative or winter) in most of the material observed in 1982. Nevertheless, good spring types were also detected. Those lines having the longer seeding to heading period appear to escape the disease more often. No studies on the nature of the resistance (genetic or escape mechanism) are currently under way. Regional surveys are conducted each year throughout the wheat areas to evaluate the distribution and severity of Septoria spp.

Table 1.--Lines with good resistance to Septoria tritici from yield trials at Hidango Experiment Station 1981-82.

Line and pedigree	Leaf blotch[1]		Days to heading[2]	
	1981	1982	1981	1982
Aurifen (spring check)	4	9	112	127
KVZ-GVxTito"S", CM 30817-C-10Y-2M-1Y-OM-4Ptz-OY	3	5	121	151
Veery"S", CM 33027-E-1M-11Y-OM-1Ptz-OY	2	6	120	138
Budifen	3	5	140	172
F2MBP-73-4-79	2	4	136	176
Azteca F67/Leda, T-5995-t-t-3t	3	5	135	179
Andifen (alternative, check)	3	6	137	180
Libun (land variety)	4	5	140	183
LFNx2 N 1220, WA 6153-6H-79	3	5	139	192
Mlfn/Nudif//Cgn/3/7C/Rabe, T-10204-t-3h	3	5	142	192
ATOU	-	9	-	197
Manquefen (Winter check)	2	3	154	202

[1]0-9 scale Saari-Prescott.
[2]1981 nursery sown in June; 1982 nursery sown in May.

Chemical control trials were started in 1978 on a limited scale. Tests at Hidango during 1981 and 1982 permitted evaluation of yield loss and comparison between a susceptible spring cultivar, Likay INIA, and a "tolerant" or resistant facultative type, Andifen, at two levels of epidemic severity (moderate in 1981, very severe in 1982).

Broad-spectrum products, such as dithiocarbamate and triadimephon were tested in 1982. Untreated controls, two sprayings, and six sprayings were compared. Results have been encouraging. A preliminary analysis of the 1982 data is shown in table 3.

Figures show that even a tolerant cultivar like Andifen, under a strong epidemic like the one in 1982, suffers at 26% loss, indicating a relatively low threshold of tolerance, while a susceptible variety may have over 70% yield reduction.

Results have confirmed the importance of variety x fungicide treatments interaction. New products, doses, timing, and number of sprayings should be further investigated.

Work on other lines of research for the future shoud be directed to better understanding of the "escape mechanism," genetic resistance, races, or

Table 2.--Resistant lines to Septoria tritici at Quilamapu and Hidango Experiment Stations, 1982, 12th ISEPTON.

Cross and pedigree	Leaf blotch[1]		Days to heading[2] (Hidango)
	Quilamapu	Hidango	
INIA 66 (check)	5	9	119
Sonora 64 (check)	6	9	119
Veery"S", CM 33027-E-1M-11Y-0M-2Ptz-0Y-2Ptz-0Y	0	5	131
Veery"S", CM 33027-E-1M-9Y-0M-1Ptz-0Y	0	5	131
Pel.72380-Art. 71, B 13374-0M-599Y-101A-101Y-0A-1Ptz-0Y	0	4	131
KVZ-K4500. L.A.4, SWO 176-3M-1Y-10Y-1Y-1M-0Y-0Ptz	3	5	140
Barpet - Manantial 4-1-2-2-1-3-0Ptz-0Y	2	4	163

[1]Spring-sown nursery at Quilamapu (September); early fall sown at Hidango (May); 0-9 scale.
[2]Early fall sown (May).

Table 3.--Effect of fungicide treatments on grain yield and yield losses caused by Septoria tritici on two wheat varieties, Hidango, 1982.

Treatments	Varieties					
	Andifen			Likay-INIA		
	Yield q/ha[1]	Percent yield, six sprayings	Percent yield loss	Yield q/ha	Percent yield, six sprayings	Percent yield loss
Untreated check	30.8	73.2	26.8	6.1	24.3	75.7
2 sprayings	38.1	90.4	9.6	7.7	30.7	69.3
6 sprayings	42.2	100.0	-	23.2	100.0	-

[1]q=quintel=100 kilograms.

biotypes; work must be done on epidemiology and its relation with control through cultural practices to manage levels of primary inoculum in the field. Sceening of an ample scope of germplasm is to be continued, as well as use of the resistant material in the crossing programs. Depending on availability of resources, artificial inoculation techniques should be implemented both for field and greenhouse testing.

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MADARIAGA - SPEAKER (for paper by Ramirez)

Q. With spring wheat, you were saying that late planted ones escape disease. With winter wheat, you said that by planting early you can also escape disease. I don't understand.

A. With the first case, spring wheat planted as late as August or September (the normal planting date), escape refers to an environmental effect which is not favorable to the fungus. Even susceptible wheats do not show attack. Winter wheats go through a favorable environment for Mycosphaerella but also "escape" from the fungus by a combination of plant habit and an unknown mechanism of tolerance and/or resistance.

Q. Ballantyne: Is it possible to get details of the pedigree of the particular Veery that you reported showing some resistance?

A. Yes, it is Veery "s," CM 33027-E-1M-11Y-0M-2Ptz-0Y-2Ptz-0Y.

Q. Haven't you found any winter wheats that are susceptible? I see your point there is inherent disease, but have you found any that are essentially very susceptible?

A. Yes, but using the scale 0-9, it is difficult to discriminate susceptibles from resistants. Usually in our conditions, it is possible to get 6 and 7 readings on some winter genotypes; however, these readings do not have any relation later on with a yield decrease. It means that maybe these winter genotypes are not very resistant, but our pathogen population is not able to overcome this low resistance so far.

OCCURENCE AND IMPORTANCE OF SEPTORIA NODORUM AND S. TRITICI IN THE ANDEAN COUNTRIES OF SOUTH AMERICA

H. J. Dubin[1]

In the course of my pathology and breeding work in the Andean countries of Ecuador, Colombia, Peru, and Bolivia, I have become concerned with the common occurrence of leaf spotting diseases in wheat breeding nurseries as well as grower fields in highland areas (about 2500-3300 masl). Over the last 2 years, observations indicated an increase in these types of symptoms probably associated with increased rainfall in the highlands. Leaf spots have not traditionally been considered important diseases on wheat in the Andes, probably due to the fact that yellow rust, Puccinia striiformis West., was so devastating previously. Now that good resistance is available for yellow rust, some of the less obvious diseases such as the leaf spots and BYDV have become more discernible and may be causing significant losses. Other reasons for the previous lack of concern about these types of diseases are probably due to the fact that the Andean Region has few cereal pathologists, the agricultural system is a subsistence type with very small holdings, and the few extension agents that work in small grains have minimal pathology training.

It was of interest to determine which, if any, pathogens were associated with the range of symptoms observed in the different areas as a possible first step in deciding if they should be dealt with in the various breeding programs in the region.

Collections of wheat leaf samples were made intermittently in breeding nurseries as well as grower fields in 1981 and 1982 in some of the wheat areas of Colombia, Ecuador, Peru, and Bolivia. Over the 2-year period, 109 leaf collections were made and 19 percent contained either Septoria tritici or S. nodorum as the predominant known pathogens. Twenty percent had Helminthosporium tritici-repentis as the predominant organism. Since the sample was small and biased, it is difficult to say which Septoria species is more important at this stage. Continuing observations throughout the region would indicate that S. tritici has predominated up to now. However, it is important to note that these fungi generally occur in complexes and it is common to encounter the Septorias together on the same plant as well as H. tritici-repentis which is probably just as important or more so than either Septoria species. All three of the above pathogens are found in the four countries. Less commonly, one finds S. avenae triticea and rarely a Phaeoseptoria species involved in these complexes. In Ecuador, Fusarium nivale may occur as a foliar blight in complex with the above fungi or alone. Soil problems commmon to the highlands appear to contribute to the blight symptoms observed.

In Ecuador, where wheat stubble has been examined, it has been common to find the teleomorph of S. nodorum (=Leptosphaeria nodorum Müller) located principally in the stem nodes. Since stubble is generally plowed under, it is doubtful that the teleomorph plays an important epidemiological role.

Microscopic examination of the aforementioned collections indicated that conidial dimensions generally fit published descriptions, for example, S. tritici macroconidia = 40 - 72 um x 2.0 um, 2 - 7 septa, and S. nodorum conidia = 15 - 30 um x 2.5 um, 1 - 3 septa.

Wheat production in the Andes is presently considered of secondary importance, and it is doubtful that much pathology research will be done in the future. Thus, breeders will have little data on which to base their breeding priorities. The simplest and most logical breeding approach at this stage is to continually select plants with the healthiest leaves.

DUBIN - SPEAKER

Q. Can you elaborate on your comment about predisposing factors?

A. We have one basic factor, acid soils that bind phosphorous, and predispose the plants to various problems of vigor, but also foot rot.

The other factor that I'm convinced is causing problems is UV light. The high UV light at these elevations is quite severe, and we do get burning. Most of the time, we will eliminate this genotype, but every so often susceptible genotypes get through, and you will find these kinds of plants also appear to be more susceptible.

Another phenomenon we see is very good resistance to leaf rust and yellow rust and on resistant lesions of yellow rust, infections by Septoria tritici.

Q. Fehrmann: Today I heard you mention several times Ascochyta. Are they common?

A. It's a fungus I just picked up when we had attacks of Septoria.

Q. Fehrmann: Are they pathogenic?

A. Some of the Ascochyta spp. can be pathogenic, but they're weak pathogens. They cause damage by and of themselves, but also are strong saprophytes and will come in with a complex.

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WORK ON SEPTORIA NODORUM BERK. IN THE INSTITUTE FOR BREEDING AND PRODUCTION OF FIELD CROPS, ZAGREB

Koric Bogdan[1]

By introducing semidwarf and dwarf wheats into production, a severe attack of leaf and head disease, known by the common name Septoria complex, was noticed. Some years its effect on quality and yield was considerable. Studying the frequency of each of the diseases, from the group Septoria complex, it was decided that the work on investigating Septoria nodorum Berk. should be started in the institute. A team consisting of a phytopathologist (Korić Bogdan) and a breeder (Rade Mlinar) was formed to create resistant or tolerant wheat varieties suited to our growing conditions. After specialization at the Swiss Federal Research Station for Agronomy with M. P. and A. A. Bronimann in 1980. laboratory work on S. nodorum fungus was started with success. Certain modifications in the method of work taken over were inevitably done because of the specific facilities at our disposal in the Institute.

The work started in the spring of 1981 with collecting leaves and spikes infected with S. nodorum. That year, 55 samples of infected leaves and spikes of different varieties and from different locations were collected. S. nodorum was determined on 17 samples (31%) mainly from spikes. The re-isolation on potato dextrose agar (PDA) was successful on 13 samples. Having studied the behavior of isolates on PDA, seven of them were picked out to be used for making inoculum (growing on sterile grain) for artificial infection in the field, which was carried out in 1982.

In 1982, 65 samples of infected leaves and spikes were collected. S. nodorum was found on 10 samples (15.4%), also mostly on spikes. Reisolation was successful on eight samples, and after studying the isolate on PDA, five samples were chosen to be used for making inoculum needed for artificial infection in the field and were reinoculated on sterile wheat grain.

Several leaf samples with no S. nodorum were found to be infected with S. tritici Rob. ex. Desm., the rest were saprophytes among which Alternaria spp. dominated. Beside saprophytes (Alternaria spp. in most), Fusarium graminearum was also determined on spike samples, which were found free from S. nodorum.

Inoculum for artificial infection in the field, in the adult stage, and in the greenhouse (later in a cold chamber) at the stage of seedling was successfully made. As a substrate for growing and developing fungus, I used wheat grains prepared by the standard method and sterilized in the autoclave.

Artificial infection was first carried out in the greenhouse with plants at the stage of seedlings, with cvs. 'Kopara', 'Zlatna Dolina', 'San Pastore', and 'Little Club', to be continued with the selected assortment. Success was incomplete because we were not in a position to control conditions like temperature, humidity, light, and other factors in the greenhouse. Therefore, we had to change the place of testing. Further tests were carried out in the cold chamber (with constant temperature 8°-10°C), which served well for growing S. nodorum on PDA on sterile grain. Light was constant (fluorescent tubes). The pots with artificially infected plants were covered with transparent plastic bags, 30 x 40 cm. In this way, the relative humidity needed for a successful infection, was achieved. The seedlings remained under these conditions until rating. Plastic bags can be removed before rating takes place, providing full attention is paid to regular watering. The symptoms of the attack usually appear after 5 or 6 days, and the rating can be taken after 8 to 12 days. The rating was taken according to the accompanying scale (fig. 1). The check plants underwent the same procedure, apparently with no evident changes. This method turned out to be very successful. As a check, I reisolated the fungus from the infected leaves, which proved to be S. nodorum originally used for artificial infection.

As check varieties for resistance and susceptibility, I chose the ones that have so far proved in my tests to be good for that purpose or the varieties

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Figure 1.--Scale for appraising Septoria nodorum Berk. in seedling stage.

internationally used as checks. Up to now I have been using the following check varieties:

'Kopara'--excellent as a susceptible cultivar in the seedling and adult stage.

'Zlatna Dolina'--susceptible in the seedling and adult stage; in Yugoslavia, it is a standard for yielding.

'Triticum timopheevi'--standard for resistance in seedling; however, in the adult stage this trait has not been completely tested.

'Hadden' and 'Fortuna'--international check varieties; have not been tested enough under our conditions.

Artificial infection with S. nodorum in the adult stage in the field, which was carried out in 1982, was successful. The analysis of the results obtained is in progress.

In field investigations, the plan of work was comprised of the study of the effect of S. nodorum on the weight of 1000 grains, hectolitre weight and production per spike, as well as finding sources of resistance or tolerance for the area investigated, which could serve in breeding work for developing resistant or tolerant varieties. We also planned to study the spreading of the disease through grains in certain selected varieties. All the observations and the results obtained will be published after a close analysis.

I would like to acknowledge the assistance of my colleagues throughout the world, who in any way helped me in my efforts to master the problems of the S. nodorum fungus.

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IDENTIFICATION AND PREVALENCE OF SEVERAL WHEAT LEAF SPOT DISEASES

Robert M. Hosford, Jr.[1]

Brown, tan, yellow, and white leaf spots on wheat are being caused by several fungal and bacterial pathogens, acting individually and in complexes. The diseases these pathogens cause can overlap in appearance, and in complexes they are often mistakenly attributed to one sporulating pathogen. Nonsporulating pathogens can often be present and may cause the greatest damage. The following is a comparison of some of these pathogens, the overlapping symptomotology of the diseases they cause, the identification of the pathogens, and their relative importance (see also table 1).

Four fungal pathogens cause irregular yellow to light-brown spots on differing cultivars, with the most susceptible cultivars being spotted following a 24-hour postinoculation wet period and more resistant cultivars requiring 48 to 72 hours. These pathogens are identified by their spore stages in older lesions, straw, and stubble or by their isolation from spots and sporulation on culture media, such as potato dextrose agar (PDA). These four fungi have caused only minor spotting, possibly due to the long wet period requirement. Their names are Platyspora pentamera (Karst.) Wehm., which has not sporulated in culture (3); Phoma glomerata (Corda) Wr. & Hochapf., which has been associated with mycotic diseases of man (4); Leptosphaeria herpotrichoides de Notaris, once thought to be the cause of foot rot of wheat (5, 15); and Leptosphaeria microscopica Karst., anamorph Phaeoseptoria urvilleana (Speg.) Sprague (6).

Bipolaris sorokiniana Shoemaker, teleomorph Cochliobolus sativus (Ito & Kurib.) Drechs. ex Eastur, causes "Spot Blotch" (small to moderate sized dark leaf spots), and root rot, and is one major cause of black point of grain. It is identified by its conidia produced in the lesions, straw and stubble; if spores do not develop on lesions, placing the lesions in moist chambers or on PDA for 1 to 7 days will induce sporulation. This fungus causes minor to major leaf spotting in many wheat growing regions of the world. Its lesions are often mingled among those caused by other leaf spotting pathogens (7, 10, 11, 14, 15).

Pyrenophora tritici-repentis Died., anamorph Drechslera tritici repentis (Died.) Shoem., causes "Tan Spot," whose symptoms range from 1) small dark spots (particularly where resistance is evident) similar to Spot Blotch; to 2) larger, coalescing tan to brown spots, containing small, dark sites of infection and often with yellow borders; to 3) dark spots surrounded by very large yellow areas, covering much of the leaf. Severely spotted leaves soon turn totally brown. This fungus is becoming recognized as a major leaf spotting pathogen of wheat worldwide, whose nonsporulating spots have been confused with similar tan-colored spots caused by Septoria species. Its smaller, darker spots have been confused with Spot Blotch. It can be identified by its conidiophores and conidia in older lesions, or, if sporulation is not occurring, through inducing sporulation by placing the lesioned leaves in moist chambers at 15°C for 12 hours of light followed by 12 hours of dark. If saprophytes grow too abundantly on spots in the moist chambers, this fungus can be isolated Septoria tritici Rob. in Desm., teleomorph Mycosphaerella graminicola (Fuckel) Sand., and Septoria nodorum (Berk.) Berk., teleomorph Leptosphaeria nodorum Müller, are recognized and Septoria avenae Frank f. sp. triticea T. Johnson, teleomorph Leptosphaeria avenaria Weber f. sp. triticea T. Johnson, is becoming recognized as major, worldwide causes of leaf spotting of wheat. These Septorias cause yellow to brown leaf spots similar to and often mixed with the tan to brown spots caused by P. tritici repentis. In Septoria spots on leaf blades pycnidia are formed, and on many, but not all wheat varieties, the centers of these Septoria spots turn gray. Identification of these Septoria diseases is based on conidia morphology and measurements. However, in the field, the more densely pycnidia filled, narrower lesions of S. tritici can often be used to distinguish this from lesions on modified V8 agar, where it can be induced to sporulate using the above light/dark period. In North Dakota wheat fields, if tan to brown spots with small dark centers and yellow borders on leaf blades have appeared before the boot stage of wheat development or have not developed Septoria pycnidia by late July and the late milk stage of wheat growth, they have been determined to be Tan Spot. These symptoms have been used to separate Tan Spot and Septoria spotting in the field. Workers should check the sporulation from spots in their areas in the laboratory before using these symptoms as identification, since nonsporulating spots caused by Septorias and/or other fungi might cause confusion. The P. tritici-repentis sexual and asexual spore stages are abundant on weathering wheat straw and stubble in many areas (1, 7, 10, 11, 15).

Septoria from the other two. Septoria nodorum also causes "Glume Blotch" in some climates. Where the yellow to brown spots have no sporulation, these pathogens, like P. tritici-repentis may be induced to sporulate in moist chambers or can be isolated from the spots on artificial media. Some isolates of S. avenae f. sp. triticea have not sporulated in culture but have produced the white mycelium and tan color (seen through the bottom of the petri plate) of sporulating isolates (1, 2, 7, 10, 11, 14, 15).

Ascochyta tritici Hori & Enjoji causes "spindle-shaped ashy-brown to ashy-white lesions" (13). It contributes to leaf damage in complexes of leaf spotting diseases but is not itself a major disease (7, 13, 15).

The three Septorias, B. sorokiniana, and P. tritici-repentis have appeared singly and in varying complexes worldwide, causing leaf spotting related to major yield losses (1, 7, 10, 11, 14, 15). The other fungi may contribute to losses in extremely wet years (3, 4, 5, 6, 7, 12, 13, 15).

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Table 1.--Several wheat leaf spot diseases.

Symptoms	Common name	Pathogen
Irregular yellow to light-brown spots	Platyspora leaf spot	Platyspora pentamera
	Phoma spot	Phoma glomerata
	None	Leptosphaeria herpotrichoides
	None	Leptosphaeria microscopica
Small to moderate dark leaf spots	Spot blotch	Bipolaris sorokiniana
	Tan spot	Pyrenophora tritici-repentis
Large tan to brown spots often with yellow borders		
1. With a small dark site of infection	Tan spot	Pyrenophora tritici-repentis
2. With pycnidia and often graying centers		
a. Graying centers narrow	Septoria tritici spot	Septoria tritici
b. Graying centers wider	Septoria nodorum spot	Septoria nodorum
	Septoria avenae f. sp. triticea spot	Septoria avenae f. sp. triticea
	Ascochyta leaf spot	Ascochyta tritici
Very large yellow areas with small dark sites of infection	Tan spot	Pyrenophora tritici-repentis
Brown streaks	Brown streak	Xanthomonas campestris pv. undulosa
Bleached white to tan streaks to blotches	Bacterial leaf necrosis	Pseudomonas syringae pv. syringae
	White blotch	Bacillus megaterium pv. cerealis

Bacteria are causing white, yellow, tan, or brown spots on wheat leaves that can be distinguished from fungal spots. "Brown Streak" of the leaves and "Black Chaff" of the head caused by Xanthomonas campestris pv. undulosa Smith, Jones and Reddy have been evident in wheat growing areas for many years, but in recent years Brown Streak has been pronounced. These long, narrow, often glistening, jagged, brown streaks, parallel to the length of the leaf can usually be distinguished from other diseases (9). However, Pseudomonas syringae pv. syringae Van Hall has been isolated from some brown streaks (12, and J. A. Otta, personal communication). This latter bacterium causes a white to tan leaf streaking and blotching on wheat called "Bacterial Leaf Necrosis." In North Dakota, in addition to these two bacterial diseases, a third disease called "White Blotch", caused by Bacillus megaterium de Bary pv. cerealis Hosford, is appearing as a bleached white to light-tan streaking and blotching on numerous wheat cultivars. This disease is similar in appearance to Bacterial Leaf Necrosis, although it begins as transient, small, pale-yellow to white spots, while Bacterial Leaf Necrosis begins as transient, green, wet spots. Distinct varietal differences in resistance to Bacterial Leaf Necrosis and White Blotch occur (8). The diseases caused by the three bacteria do not display asmall dark site of infection or yellow border, as some of the fungal spots do, and tend to be in streaks and be more sharply edged than fungal spots. These characteristics and white bleaching are used to distinguish the bacterial spots from fungal spots. White Blotch can be oval shaped. All three of these bacteria are causing severe leaf damage on differing wheat cultivars in various areas, but the geographical and varietal extent of the damage they cause is undetermined. Isolation, identification, and establishing pathogenicity of each bacterium are needed to distinguish and evaluate each of these bacterial diseases.

The above are several (but not all) wheat leaf spot diseases whose overlapping symptoms confuse their identification and individual importance. Each disease and each complex of diseases needs to

be distinguished and evaluated for distribution, related losses, varietal resistance, epidemiology, chemical and cultural controls, influence of environment, and other factors.

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HOSFORD - SPEAKER

Q. Tomerlin: You mentioned that you had some varieties resistant to a 72-hour dew period. Do you know if that works 72 hours over a 4- or 5-day period or does the dew period have to be continuous?

A. Our attempts to clear up questions of the wet period have been with mixed results. I'm hoping that someone else will also work on it.

Q. Dubin: Could you clarify why, when you discussed Bacillus magaterium, you said I'm going out on a limb on this?

A. I was taught as a child that anybody who said Bacillus spp. caused disease was suspect. In the world literature, there are only about five or six articles on Bacillus spp. causing spotting or disease and none of those are confirmed. There is literature on potato disease indicating the potatoes were under extremely high temperatures. This study has confirmed Bacillus species as the cause of the disease. So, this doesn't appear to be very much of a pathogen.

Q. Dubin: Is your black chaff disease similar to head melanism?

A. I am assuming head melanism is the same thing we call physiological black chaff, which is genetic. This is bacterial black chaff caused by X. translucens. In North Dakota, we have at least two types of black chaff of the head, the bacterial and the physiological.

Q. Ramsey: When you're looking at standard wetness periods that affect some of these diseases, what sort of light would you require?

A. The moist chamber are in a greenhouse with a very thin flexible piece of plastic over them so they are getting bright light conditions. We try to do most of our work in the winter, fall and spring and not in the summer because we have to prevent a heat trap. Usually, there is quite bright light both from the sun and from artificial lighting in the greenhouse. That was one of the first questions people asked me: "Are you putting these things in the dark and is the dark causing damage?"

ALTERNATIVE HOSTS AND OVERSEASONING OF SEPTORIA NODORUM

J. M. Krupinsky[1]

Septoria nodorum (Berk.) Berk. (perfect state = Leptosphaeria nodorum Müller), a pathogen on wheat (Triticum aestivum L.), has been reported on a wide range of gramineous hosts (7). The present study reports on the pathogenicity of various isolates of S. nordorum on forage grass species.

Ten cultures of S. nodorum were isolated, maintained on V-8 juice agar, and used for inoculum as previously reported (3). The five isolates from Agropyron spp. used in earlier studies (2, 3) were used in the present study. These isolates were from intermediate wheatgrass [A. intermedium (Host.) Beauv.], western wheatgrass (A. smithii Rydb.), diploid crested wheatgrass [A. cristatum (L.) Gaertn.], tetraploid crested wheatgrass [A. desertorum (Link) Schult.], and a hybrid of quackgrass [A. repens (L.) Beauv.] X A. desertorum. One isolate from wild barley (Hordeum jubatum L.), two isolates from spring wheat, and two isolates from smooth bromegrass (Bromus inermis Leyss.) were also used. Alternative hosts were grown in a glasshouse with a 12-hour photoperiod and temperatures of 24±4°C for the light cycle and 13±4°C for the dark cycle. After inoculation, the plants were maintained in a high humidity chamber for 48 hr (4) and then moved to a glasshouse bench. Leaves were visually assessed twice between 7 and 12 days after inoculation for percent necrosis, number of lesions, and lesion size. Percent necrosis ratings were recorded as the percentage of necrotic leaf blade tissue. The number of lesions on the leaves was rated as none (0), very few (1), few (2), intermediate (3), and numerous (4). Lesions were rated according to their size as none (0), very small (1), small (2), medium (3), and large (4). In order to confirm pathogenicity on alternative hosts, eight leaf samples from each grass species inoculated with each of five isolates were plated on water agar after surface sterilization. The five isolates included two isolates which were originally obtained from spring wheat and one each from diploid crested wheatgrass, wild barley, and smooth bromegrass. In addition, S. nodorum was reisolated from all grass species inoculated with three isolates originally obtained from spring wheat, diploid crested wheatgrass, and wild barley to determine if isolates were still pathogenic on wheat after one passage through an alternative host.

Isolates from wheat, Agropyron spp., smooth bromegrass, and wild barley could not be differentiated from one another when considering symptoms on alternative hosts. In general, alternative hosts expressed symptoms when inoculated with all 10 isolates of S. nodorum from different sources (table 1). S. nodorum was present on all alternative hosts and on 98% of the leaf samples that were plated on water agar (table 1). Thus, these isolates, originally from different hosts, were pathogenic on all alternative hosts. The three isolates, originally obtained from wheat, diploid crested wheatgrass, and wild barley, were each reisolated from all 24 alternative hosts inoculated. Symptoms were produced on four cultivars of wheat ('Fortuna', 'James', 'Angus', and 'Kitt') when they were inoculated with each of the 72 reisolated cultures. Thus, these three isolates were still pathogenic on wheat after passage through each alternative host.

Based on necrosis produced on the alternative hosts by all 10 isolates, the (Elymus spp. were the most severely infected (table 1). Basin wildrye (Elymus cinereus Schribn. and Merr.), Siberian wildrye (E. sibericus L.), and Altai wildrye (E. angustus Trin.) have less overall symptoms than Fortuna, a susceptible wheat cultivar, but more than Angus, a resistant cultivar. Only seven other alternative hosts had overall necrosis greater than 10%: diploid crested wheatgrass, beardless wheatgrass [A. spicatum f. inerme (Scribn. and Sm.) Bettle], bluebunch wheatgrass [A. spicatum (Pursch) Scribn. and Smith], beardless wildrye (Elymus triticoides Buckl.), mammoth wildrye (E. giganteus Vahl.), blue grama [Bouteloua gracilis (H.B.K.) (Griffiths)], and green needlegrass (Stipa viridula Trin.). The 14 remaining alternative hosts had overall necrosis averaging less than 10%, ranging from 1 to 9% necrosis. Eleven alternative hosts with 6% or less necrosis were considered the most resistant: intermediate wheatgrass, western wheatgrass, big bluestem (Andropogon gerardii Vitm.), little bluestem (Andropogon scoparius Michx.), sand bluestem (Andropogon halli Hack.), creeping foxtail (Alopecurus arundinaceus Pair.), orchardgrass (Dactylis glomerata L.), indian grass [Sorghastrum nutans (L.) Nash.], prairie sandreed [Calamovilfa longifola (Hook.) Scribn.], smooth bromegrass, and switchgrass (Panicum virgatum L.).

In general, the number of lesions produced on alternative hosts by isolates of S. nodorum was low. The Elymus spp. had the greatest number of lesions of the alternative hosts (table 1). Eleven alternative hosts had very few to few lesions. The remaining nine alternative hosts had ratings less than 1, very few lesions: intermediate wheatgrass, Russian wildrye (Elymus junceus Fisch.), little bluestem, creeping foxtail, sand bluestem, orchardgrass, prairie sandreed, bromegrass, and switchgrass.

Eight alternative hosts had overall lesion size ratings over 2, small: diploid crested wheatgrass, beardless wheatgrass, bluebunch wheatgrass, basin wildrye, Altaia wildrye, Siberian wildrye, mammoth wildrye, and reed canarygrass (Phalaris arundinacea L.) (table 1). Except for reed canarygrass, this rating was comparable to the rating of Fortuna. Thirteen alternative hosts had overall lesion size ratings from 1, very small, to 2, small. Only two alternative hosts were rated less than 1 for lesion size: little bluestem and prairie sandreed.

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Table 1.--Pathogenicity of Septoria nodorum isolated from Triticum aestivum, Agropyron spp., Bromus inermis, and Hordeum jubatum.

Host	Percent necrosis	Number of lesions [1]	Lesion size [2]	S. nodorum present [3]
Agropyron cristatum [8]	[4]11	[4]1.2	[4]2.2	40/40
Agropyron desertorum [8]	9	1.1	1.6	36/40
Agropyron spicatum f. inerme	16	1.4	2.5	40/40
Agropyron intermedium [8]	6	.9	1.2	40/40
Agropyron spicatum [5]	15	1.7	2.3	38/40
Agropyron smithii [5 8]	5	1.5	1.7	40/40
Elymus angustus [10]	17	1.8	2.3	40/40
Elymus cinereus [10]	21	2.0	2.3	40/40
Elymus sibericus [5]	21	2.2	2.6	40/40
Elymus triticoides	11	1.7	1.8	40/40
Elymus giganteus [5 9 10]	14	2.0	2.4	39/40
Elymus junceus [5 10]	7	.7	1.0	33/40
Andropogon scoparius	1	.7	.5	39/40
Andropogon gerardii	3	1.2	1.4	39/40
Bouteloua gracilis	13	1.2	1.5	38/40
Alopecurus arundinaceus	3	.8	1.0	40/40
Andropogon hallii	3	.8	1.1	38/40
Stipa viridula [5 6]	12	1.0	1.2	40/40
Sorghastrum nutans	4	1.0	1.4	39/40
Dactylis glomerata [7]	5	.6	1.3	40/40
Calamovilfa longifolia	2	.4	.5	40/40
Phalaris arundinacea	7	1.3	2.1	39/40
Bromus inermis [7 10]	4	.9	1.5	40/40
Panicum virgatum	3	.8	1.1	38/40
Triticum aestivum-Angus [5 6 7 9]	8	1.3	1.8	
Triticum aestivum-Kitt [5 6 7 9]	10	1.4	1.6	
Triticum aestivum-Fortuna [5 6 7 9]	29	3.1	2.8	
L.S.D. (0.05) for all hosts	6	.6	.6	

[1]0 = no symptoms, 1 = very few, 2 = few, 3 = intermediate, and 4 = numerous.

[2]0 = no symptoms, 1 = very small, 2 = small, 3 = medium, and 4 = large.

[3]First number indicates the number of leaf pieces on which the fungus was present; the second number is the number of leaf pieces that were plated. 8 leaf samples from each of 5 inoculations were plated on water agar.

[4]Each value is the average of 40 observations (20 inoculations, 2 observations/inoculation); 8 observations for isolates from T. aestivum, 20 observations for isolates from Agropyron spp., 8 observations for isolates from B. inermis, and 4 observations for 1 isolate from H. jubatum.

[5]Species previously reported as a host (7).

[6]Species previously reported as a host (6).

[7]Species previously reported as a host (5).

[8]Species previously reported as a host (2,3).

[9]Species previously reported as a host (1).

[10]S. nodorum recently isolated (J. M. Krupinsky, unpublished).

After the inoculation studies were completed, S. nodorum was isolated from field collections of Altai wildrye, basin wildrye, Russian wildrye, and mammoth wildrye (table 1). Considering the high level of symptom expression produced on Elymus spp. in this study, the presence of S. nodorum on Elymus spp. is to be expected. Apparently, S. nodorum has a broad host range. S. nodorum has the ability to infect a large number of alternative hosts and isolates are still pathogenic on wheat after passage through alternative hosts. Thus, S. nodorum could overseason on the alternative hosts. Certain alternative hosts, such as smooth bromegrass common along roadways and found in close proximity to wheat fields, could serve as a source of inoculum and genetic variability for the fungus.

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RESISTANT WHEAT TESTED WITH SEPTORIA NODORUM ISOLATED FROM TRITICUM AESTIVUM, HORDEUM JUBATUM, BROMUS INERMIS, AND AGROPYRON SPECIES

J. M. Krupinsky[1]

Septoria nodorum (Berk.) Berk. (perfect state = Leptosphaeria nodorum Müller), a pathogen on wheat (Triticum aestivum L.), has been reported on a wide range of gramineous hosts (6). Isolates of S. nodorum from wheat can infect alternative hosts (5, 7), and isolates from alternative hosts can infect wheat (2, 7). The effect on resistant wheat of 10 isolates of S. nodorum that had been obtained from eight different hosts is reported in this paper.

Ten cultures of S nodorum were isolated, maintained on V-8 juice agar, and used for inoculum as previously reported (2). The five isolates from Agropyron spp. used in earlier studies (1, 2) were used in the present study. These isolates were from intermediate wheatgrass [A. intermedium (Host.) Beauv.], western wheatgrass (A. smithii Rydb.), diploid crested wheatgrass [A. cristatum (L.) Gaertn.], tetraploid crested wheatgrass [A. desertorum (Link) Schult.], and a hybrid of quackgrass [A. repens (L.) Beauv.] X A. desertorum. One isolate from wild barley (Hordeum jubatum L.), two isolates from spring wheat, and two isolates from smooth bromegrass (Bromus inermis Leyess.) were also used. All 10 isolates of S. nodorum were used to inoculate two groups of wheat cultivars. Inoculations were conducted on 12 spring wheat cultivars, group 1 (table 1), and later on 17 cultivars, group 2 (table 1), which included 13 wheat cultivars which have been reported as resistant to S. nodorum (3).

Wheat plants were grown in a glasshouse with a 12 hour photoperiod and temperatures of 24±4°C for the light cycle and 13±4°C for the dark cycle. After inoculation, the plants were maintained in a high humidity chamber for 48 hr (4) and then moved to a glasshouse bench. Leaves were visually assessed twice between 7 and 12 days after inoculation for percent necrosis, number of lesions, and lesion size. Percent necrosis ratings were recorded as the percentage of necrotic leaf blade tissue. The number of lesions on the leaves was rated as none (0), very few (1), few (2), intermediate (3), and numerous (4). Lesions were rated according to their size as none (0), very small (1), small (2), medium (3), and large (4). To confirm pathogenicity of the various isolates on wheat, leaf samples from each wheat cultivar inoculated were plated on water agar after surface sterilization.

All 10 isolates of S. nodorum were pathogenic on wheat. Necrosis and lesions were found on all cultivars inoculated. S. nodorum was present on all 12 cultivars of wheat in group 1 when leaf samples were plated on water agar (table 1). The isolates of S. nodorum from wheat caused significantly more necrosis overall at 27% compared with 6, 6, and 7% for the isolates from Agropyron spp., smooth bromegrass, and wild barley, respectively (table 1). The greatest number of lesions was associated with the isolates from wheat, which resulted in an overall rating of 3.0 compared with 1.3, 1.3, and 1.0 for the isolates from Agropyron spp., smooth bromegrass, and wild barley, respectively. Also, the largest lesions were associated with isolates from wheat, which resulted in an overall rating of 3.6 compared with 2.1, 2.0, and 2.0 for the isolates from Agropyron spp., smooth bromegrass, and wild barley, respectively. As previously found with isolates from Agropyron spp. (2), the isolates from smooth bromegrass and wild barley are less virulent than isolates from wheat.

Overall 'Fortuna', 'James', 'Eureka', 'Waldron', and 'Alex' had the greatest amount of necrosis in the first group of wheat cultivars (table 1). All other cultivars had necrosis ranging from 1 to 12% overall. 'Angus', 'Kitt', 'Butte', 'Len', and 'Sinton' were the most resistant (table 1). Fortuna and James had the greatest number of lesions with an overall average of 3.0 and 3.4, respectively. The fewest lesions occurred on Angus (0.6), Kitt (0.9), Butte (1.1), 'Coteau' (1.2), and Sinton (1.0). The cultivars with the largest lesions were Fortuna (3.4), James (2.9), Len (3.1), Alex (2.6), 'Olaf' (3.2), Eureka (3.1), and Waldron (3.6). The cultivars with the smallest lesions were Angus (0.8), Kitt (1.4), Butte (1.7), and Sinton (1.7). Apparently, the size of lesions was not restricted except on the more resistant cultivars.

S. nodorum was present on all cultivars in the second group of cultivars when leaf samples were plated on water agar (table 1). Although the isolates from wheat caused more necrosis on wheat they could not be differentiated statistically from the isolates from Agropyron spp. and smooth bromegrass (table 1). Perhaps this is due to the fact that group two contains a large number of resistant cultivars which were selected for a low level of necrosis (3). There were significantly more lesions on wheat leaves with the isolates from wheat, which resulted in an overall rating of 2.2 compared with 1.2, 1.0, and 0.8 for the isolates from Agropyron spp., smooth bromegrass, and wild barley, respectively. Also, the largest lesions were associated with the isolates from wheat, which resulted in an overall rating of 2.3 compared with 1.4, 1.1, and 0.8 for the isolates from Agropyron spp., smooth bromegrass, and wild barley, respectively. Thus, when considering lesion number and lesion size, the isolates from Agropyron spp., smooth bromegrass, and wild barley were less virulent on the resistant wheat than isolates from wheat.

Considering necrosis, the cv. James was the most susceptible cultivar tested (table 1). The susceptible cv. Fortuna, could not be differentiated statistically from N 351, 'Anderson', and 'Ziraat Fak Kutuk' on the basis of necrosis (table 1). Continuing to rate on necrosis, 'Gasta' and eight other cultivars, (Angus, Kitt, 'Hadden', 'Harvest

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Table 1.--Symptoms of Triticum aestivum inoculated with Septoria nodorum isolated from T. aestivum, Agropyron spp., Bromus inermis, and Hordeum jubatum

Cultivar	Growth habit[2]	Percent necrosis[1]: Isolates from Triticum aestivum	Isolates from Agropyron species	Isolates from Bromus inermis	Isolates from Hordeum jubatum	Overall for host	S. nodorum present[3]
Group 1							
Angus	S	6	1	0	0	2	31/40
Butte	S	30	1	3	1	9	39/40
Coteau	S	30	6	6	5	12	39/40
Fortuna	S	43	14	13	15	21	37/40
James	S	23	20	14	15	18	39/40
Kitt	S	10	1	1	1	3	40/40
Len	S	19	6	6	5	9	39/40
Alex	S	35	6	5	5	13	40/40
Olaf	S	23	7	9	8	12	40/40
Sinton	S	24	3	4	2	8	38/40
Eureka	S	45	5	7	13	18	40/40
Waldron	S	33	8	8	15	16	40/40
L.S.D.(0.05)		11	3	4	8	8	
Isolates overall		27±7	7±4	6±3	7±4		
Group 2							
Angus	S	18	7	6	2	8	8/8
Fortuna	S	35	15	20	8	20	7/8
James	S	58	48	45	30	45	8/8
Kitt	S	23	4	4	4	9	8/8
ELS 6404 -102-3[4]	S	18	12	9	5	11	8/8
Gasta[4]	W	9	2	2	0	3	8/8
Hadden[4]	W	10	8	9	5	8	7/8
Harvest Queen[4]	W	18	8	3	3	8	8/8
Michigan Amber[4]	W	30	8	8	8	14	4/4
Moking[4]	W	6	5	5	2	5	8/8
Red Chief[4]	W	13	7	4	8	8	7/8
Redhart[4]	W	30	8	4	4	12	7/8
Yamhill[4]	W	13	8	4	5	8	7/8
N 351[4]	W	30	13	15	8	17	7/8
NC 4761[4]	W	11	4	4	3	6	7/8
Anderson[4]	F	23	20	14	20	15	8/8
Ziraat Fak Kutuk[4]	F	35	21	14	8	20	
L.S.D. (0.05)		10	5	7	4	6	
Isolates overall		22±7	12±6	10±5	6±3		

[1]Each number for T. aestivum is the average of 4 observations (2 isolates, 1 inoculation, 2 observations per inoculation); for Agropyron spp., 10 observations with 5 isolates; for B. inermis, 4 observations with 2 isolates; for H. jubatum, 2 observations with 1 isolate; 20 observations overall (10 inoculations, 2 observations per inoculation). Percent necrosis ratings were recorded as the percentage of necrotic leaf blade tissue.

[2]S = spring, W = winter, and F= facultative.

[3]First number indicates the number of leaf pieces on which the fungus was present; the second number is the number of leaf pieces that were plated.

[4]Reported as resistant (3).

Queen', 'Moking', 'Red Chief', 'Yamhill', and NC 4761) were the most resistant.

In general, Fortuna (2.5), James (2.9), and ELS 6404-102-3 (2.4) had the greatest number of lesions. The fewest lesions occurred on NC 47612 (0.5), Gasta (0.8), Hadden (0.6), Harvest Queen (0.9), Michigan Amber (0.8), Moking (0.7), Red Chief (0.7), and N 351 (0.7). ELS 6404-102-3 (2.3), Fortuna (2.2), James (2.0), Anderson (1.8), and Ziraat Fak Kutuk (1.9) had the largest lesions. N 351 (0.7), NC 4761 (0.7), Gasta (1.0), Hadden (0.9), Harvest Queen (1.2), Michigan Amber (1.1), Moking (0.9), and Red Chief (0.9) had the smallest lesions.

In general, cultivars of wheat, selected for resistance to S. nodorum with a mixture of isolates from wheat, were also resistant to isolates of S. nodorum isolated from the other grasses. Even though isolates from nonwheat hosts were pathogenic to wheat, they were of lower virulence than the wheat isolates. Isolates from wild barley and smooth bromegrass would probably be the most common isolates from alternative hosts encountered by wheat plants in the northern Great Plains because of the wide distribution of these two hosts. Smooth bromegrass is commonly found along roadways and found in close proximity to fields of wheat. Wild barley is a common weed found throughout the northern Great Plains. Considering that isolates from these hosts are less virulent than isolates from wheat, wheat selected for resistance with isolates of S. nodorum from wheat would be resistant to isolates from alternative hosts. Thus, when screening wheat for resistance to S. nodorum, isolates from wheat should continue to be used.

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KRUPINSKY - SPEAKER

Q. Peter Scott: You commented on variation in pathogenicity and you said that there wasn't much, if I understand correctly. Variation of isolates from different hosts were of similar pathogenicity on the grass hosts. You also commented on variation of resistance of the grasses, and you pointed out that there was a lot -- some grasses were more resistant than others. What you didn't comment on specifically was whether there was any interaction between the grass species and isolate groups, except by implication at the end of your talk. I think I interpreted that there was evidence of interaction to the extent of isolates from wheat showing adaptation to wheat. Could you comment on whether that is correct?

A. Yes, basically I think what you have stated is correct.

Q. Scott: So, the extent of interaction between host species and pathogen isolates is simply that isolates from wheat showed a preference for wheat. Is that correct? (YES) Otherwise, there was no host preference evident? (NO) For example, isolates from barley, I didn't pick up any particular adaptation to barley.

A. I was using one isolate from barley. I didn't have isolates from commonly grown barley cultivars.

Q. Gareth Jones: Did you find any symptoms in these grass species?

A. Yes, but some of the grasses that were very resistant had very few symptoms.

Q. Hosford: Do you find much severe Septoria nodorum spotting on flag leaves?

A. Yes, we found the leaf spotting early in the year.

Q. Fehrmann: I would like to go back to the first slide. In Germany, the Netherlands and Switzerland, several people observed specific symptoms which were not mentioned here, a specific kind of leaf blotch, which is longish. The color is more grayish, and there is a gray center.

A. I did isolate Fusarium on occasion, but I considered it to be a minor part of the complex. Maybe, Dr. Hosford has found something.

Hosford: This year, on wheat at Minot, we picked up a steely gray spot which seems to be quite different from things we have seen before. But I haven't identified it yet.

SOURCES AND IMPORTANCE OF PRIMARY INFECTION AND IDENTITIES OF ASSOCIATED PROPAGULES

F. R. Sanderson[1], A. L. Scharen[2], and P. R. Scott[3]

The first step in formulating any control strategy, is to establish the life cycle of the organism, especially the source and timing of the primary infection. With the septoria diseases, as most workers only encounter the pycnidial stages while working with the diseases during the growing season, it is generally assumed that the primary inoculum is by windblown or trashborne pycnidiospores. Conversely, because of the difficulty, even for the trained eye, in detecting pseudothecia of either Mycosphaerella graminicola (Fuckel) Schroeter or Leptosphaeria nodorum E. Müller on stubble or crop debris, there are few records of either organism in the literature. Again, it seems natural to relate the importance to the frequency of sighting. Thus, the importance of the perfect states of both species as the source of primary inoculum is being greatly underestimated.

Pseudothecia of both M. graminicola and L. nodorum develop on the dead leaf tissue of stubble, with L. nodorum also occurring on the leaf sheath. L. nodorum has also been found at Bozeman late in the growing season in lesions on green leaves. Both psuedothecia appear similar to their corresponding pycnidia with those of M. graminicola remaining within what appears to be the confines of the old lesion. Those of L. nodorum are scattered over the leaf and leaf sheath, suggesting that of the two, L. nodorum becomes saprophytic after the death of the plant.

The structures of the pseudothecia also differ. That of M. graminicola has two layers with the outer layer pigmeted, contrasting with L. nodorum, which has a single layer of up to five pigmented cells (table 1).

In the Australian wheat belt, during the growing season, there are only three types of fields. Those of the current wheat crops, those being cultivated for next year's season, and those stubble fields which were last year's crops. During favorable weather conditions, the inoculum potential, under such a system, is enormous. In New Zealand, about 10% of the wheat crop, usually those fields which were undersown with either clover or grass, are left as stubble during the winter. All stubble is removed, either by heavy grazing or by mowing by the early spring. In the United Kingdom, very few crops are left standing as stubble after harvest, yet there is very strong evidence that, even under these conditions, ascospores are the primary source of infection.

Table 2 shows the infection levels, as measured by psycnidiospore counts, made after washing the lowest leaf of 25 plants, of 17 wheat crops which were sampled near Cambridge, England, during the spring of 1980. The previous crop in each instance was determined either from crop residues or from volunteer plants.

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Table 1.--Descriptive comparisons of Mycosphaerella graminicola and Leptosphaeria nodorum.

	Mycosphaerella graminicola septoria tritici blotch	Leptosphaeria nodorum septoria nodorum blotch
Pseudothecia	On dead leaves, grouped within area of old lesion; dark brown to black.	Scattered on dead leaves and sheath. Light buff.
Pseudothecial	Two layered with outer layer pigmented.	Single layer of pigmented cells up to 5 cells thick.
Ascospores	Two celled, 10-15 um x 2-3 um.	Four celled, 19-23 um x 4 um.
Pycnidia	60-200 um, black.	160-210 um, brown.
Pycnidiospore	35-98 x 1-3 um, 3-5 septa.	15-32 x 2-4 um.
Lesions	Straw colored, irregular to rectangular, elongated between parallel veins often speckled due to prominent, numerous pycnidia.	Discolored to brown, often lens shaped, with chlorotic yellow border; central portion with pycnidia.

Table 2.--A comparison of the levels of primary infection of Septoria tritici blotch and Septoria nodorum blotch in 17 wheat crops growing in the Cambridge area, England. May 1980.

Previous crop	Numbers of spores	
	M. graminicola	L. nodorum
Wheat	0	21
Wheat	0	83
Wheat	0	278
Barley	1	3
Beans	4	56
Wheat	10	53
Wheat	14	26
Wheat	18	275
Peas	20	17
Peas	25	23
Wheat	29	27
Sugarbeet	48	433
Wheat	66	22
Wheat	204	886
Peas	304	21
Peas	556	200
Peas	1,067	136

If the primary infection had resulted from pycnidiospores produced on the crop debris of the previous wheat crop, then the highest levels of infection would have occurred in those wheat crops following wheat, with low levels of infection in the first year crops. As can be seen from table 2, the data do not support such a hypothesis, rather that the infection arose from an external source of inoculum.

The infection levels of M. graminicola appear to reflect the higher nitrogen status of the soils in those crops following peas. The infection levels of L. nodorum, it is suggested, reflects the susceptibility of the wheat cultivars to septoria nodorum blotch.

Two trials comparing different forms of cultivation gave an excellent opportunity to compare the effects of trash on the levels of the primary infection (table 3). Again, the oldest leaf from 25 plants, from each treatment, was collected and washed, and the pycnidiospores were counted. As in table 2, the infection levels suggest an external source of inoculum, which masks the local effect, if any, of infection arising from pycnidiospores produced on the trash.

When considering primary inoculum, it is extremely important to distinguish between crop debris, that which is left on the soil surface after cultivation, and crop stubble, that which is left standing after harvest. Tables 2 and 3 both refer to crop debris. In temperate regions where there is sufficient rainfall, the higher humidity at the soil surface means that crop debris remains wet for much longer periods than standing stubble. This encourages a wide range of saprophytic organisms, especially bacteria, which rapidly break down the leaf tissue, the substrate for the pseudothecia. It is suggested that the limited contribution of crop debris as a source of primary inoculum in these regions is a result of this rapid colonization of the leaf material; hence, by the time the wheat emerges in the autumn, this material has already reached the point where it is no longer a source of primary inoculum.

If wheat straw is collected at harvest, however, and stored until it is required as a source of

Table 3.--A comparison between the primary infection of wheat seedlings growing in plots with varying degrees of wheat debris remains on the soil surface. At the Norfork Agricultural Farm the area of each cultivation treatment was 100 x 25 m. The area of each treatment in the ADAS trial was 1 ha.

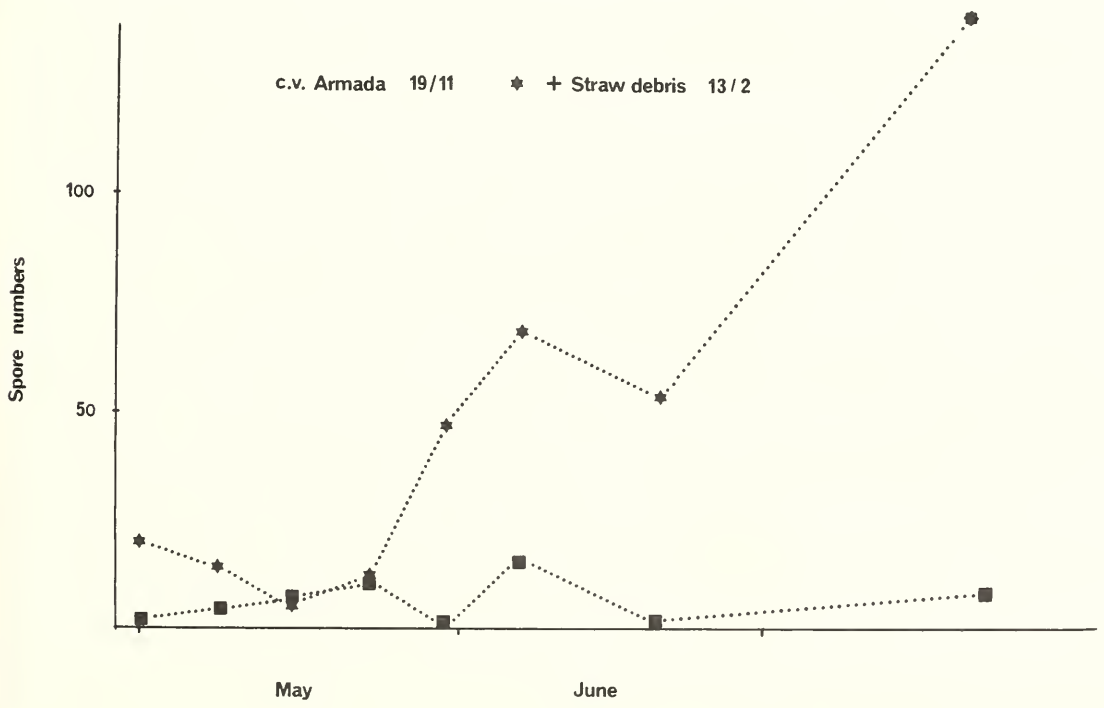
	Spore counts	
	M.graminicola	L. nodorum
Norfolk Agricultural Farm c.v. Flanders		
Convention plough (no trash)	44	23
Tined cultivation (little trash)	37	18
Direct drilled (abundant trash)	46	9
ADAS Trial c.v. Mardler + benomyl, 11 April		
Convention plough	1	188
Tined cultivation	2	109
Direct drilled	9	86

infection for inoculating plots, then this breakdown does not occur, and the pycnidia on this material will act as a source of primary inoculum.

The difference in infection levels between the two treatments in figure 1 remained clearcut, as pseudothecia would not have formed on this straw in storage and because the trial was sown in mid-November, emerging too late to be infected by airborne ascospores.

In areas such as Israel, where weather conditions appear unfavorable for the development of the perfect states and the absence of rain allows the leaf material to remain on the soil surface for long periods, and where wheat is sown directly

Figure 1.--Infection levels of Mycosphaerella graminicola in cultivar 'Armada' with and without straw debris.



into wheat trash, pycnidia are the primary source of inoculum. New infections will take place whenever the weather conditions are favorable for the liberation of pycnidiospores. Hence, multiple applications of chemicals are required to control the disease during the growing season (2).

Standing stubble on the other hand is in a much better physical position to produce and release the ascospores or pycidiospores, which act as the primary inoculum. The standing stubble, because it is predominantly dry and when wetted dries out rapidly, is not subjected to the rapid breakdown by saprophytic bacteria. The leaf material thus remains intact and a source of primary inoculum for as long as the stubble remains standing.

The type of fruiting body produced on stubble will depend on the weather conditions at the time. During milder autumn and winter conditions, pseudothecia and ascospores are produced in such places as Europe, the United Kingdom, New Zealand, and Australia. A change to pycnidial development could occur during the following spring.

DISPERSAL OF PYCNIDIOSPORES

Pycnidiospores are exuded from the mature pycnidium in the presence of moisture, with the individual pycnidiospores being forcibly dispersed by rain splash. Although Faulkner and Colhoun detected pycnidiospores 2 m above the stubble and subsequent wheat crop during rain, two conditions are likely to prevent widespread, long range dispersal of these spores: (1) If the rain is heavy, then it is likely that the scrubbing effect of the rain will wash the spores back into the crop; or (2) if the rain is light and there is a light wind, then you would expect the same effect as with aerial drift spraying. Here when the droplets are less than 100 um, they are caught up in the air currents which are moving over the surface of the crop and are effectively rolled into the crop. The result is surprisingly little downwind drift.

DISPERSAL OF ASCOSPORES

Ascospores are also released in the presence of water; hence, the points made regarding the dispersal of pycnidiospores during rain also apply to ascospores.

As can be seen from figures 2 and 3, free water to induce ascospore release is not only as a result of rain but can occur through dew, fog, and thaw after frost. It is in conditions of warming air, such as after sunrise following a dew or frost, which would create the ideal conditions of unstable rising air to carry the newly released ascospores into the air, and it is the succession of such conditions, during the autumn and early winter months, which produce the widespread uniform infection typical of high density windborne inoculum.

The evidence for rust spores from Australia frequently infecting crops in New Zealand, a distance of over 2,000 km, is overwhelming. Within one year of the two poplar rusts, and stripe rust of wheat, first being detected in Australia, all three rusts were recorded in New Zealand. There are many other examples, including two species of aphids. There is no reason to doubt that ascospores, once airborne and given the same conditions, would not travel similar international distances.

SEEDBORNE INOCULUM

There are conflicting reports in the literature on the importance of seedborne inoculum of L. nodorum in the epidemiology of septoria nodorum blotch. There is no doubt that there have been many instances when seedborne inoculum has led to yield reductions. It is certainly of major importance in the long range dissemination of the disease. However, if weather conditions are suitable for the build up of the disease early in the season, then it is likely that airborne inoculum would

Figure 2.--Ascospore release by Mycosphaerella graminicola in relation to moisture. Spore trap catches at Lincoln, New Zealand, associated with dew/a/, rain/b/, ground fog/c/, or thaw after heavy frost/d/.

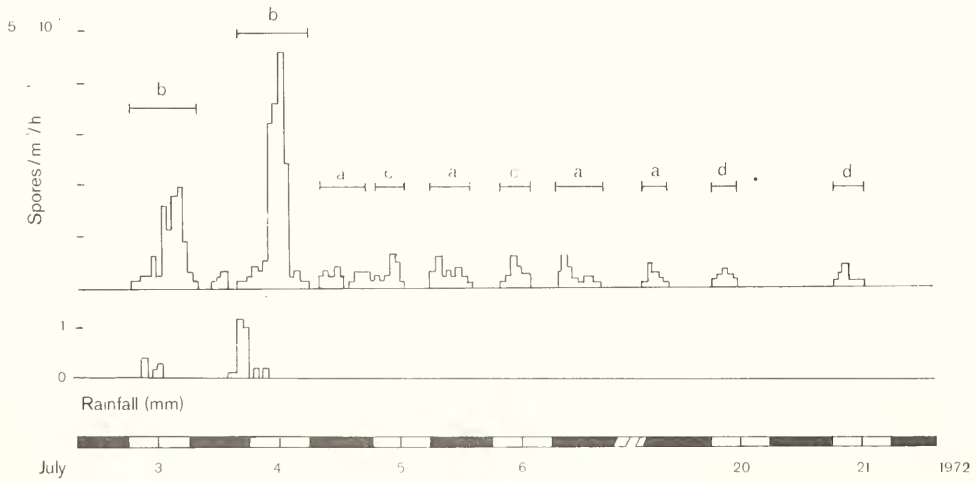
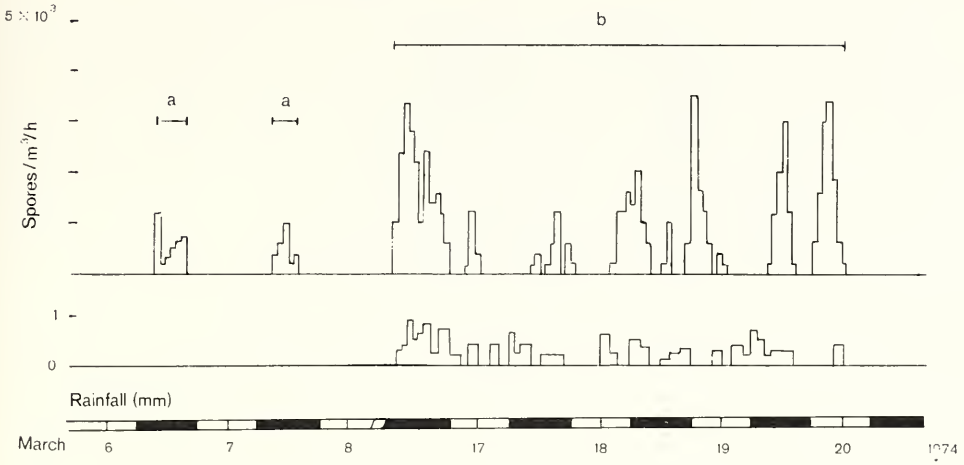


Figure 3.--Ascospore release by Leptosphaeria nodorum in relation to moisture. Spore trap catches at Lincoln, New Zealand, associated with dew/a/, rain/b/, ground fog/c/, or thaw after heavy frost/d/.



overshadow the localized foci of infection arising from the seedborne infection.

THE IMPORTANCE OF SOWING DATE ON PRIMARY INFECTION

From years of trial data and especially commercial trials, it has become apparent that sowing date is a critical factor in the establishment of primary infection within the crop in New Zealand, as delaying the sowing until the end of May or early June will guarantee little or no infection in the early spring.

IMPORTANCE OF PRIMARY INFECTION

In places such as New Zealand, Australia, and the United Kingdom, where ascospores play a major role as the primary source of inoculum, the first symptoms of septoria tritici blotch and septoria nodorum blotch appear during the early spring when the plant has reached the five-leaf stage, yet from observations of sowing date, it is likely that the infection took place in the late autumn. The importance of this early infection has been demonstrated consistantly over a number of years, for septoria tritici blotch in New Zealand, from spray trials comparing early and late infections.

In all trials, yield increases have resulted by maintaining the seedlings free of disease. As a result of this information, a disease control strategy was formulated, where benomyl was applied when the plants were at the 4 to 5 leaf stage. This is after the infection period during the late autumn and early winter, yet before the symptoms are fully expressed. This strategy is based on the premise that it is easier to control the primary infection before it becomes established, that the primary infection is more predictable, and most important of all, that the increase in yield obtained from the early control is sufficient in itself to cover the costs of application.

However, because of a major swing to the growing of the highly resistant cultivar Rongotea, the use of triadimenol (Baytan) as a seed treatment and triadimefon (Bayleton) as a spray early in the spring to control stripe rust, and a change in the winter and spring weather patterns, septoria tritici blotch has not been evident in New Zealand over the past three years. As a result, this control strategy was only tested on a limited scale.

In the United Kingdom and Europe, on the other hand, this technique has been in practice, although unwittingly, for the past decade, for as long as the MBC's (benomyl) have been used to control eye spot.

We would suggest that this is a contributing factor why septoria tritici blotch is only occasionally noted as a problem in these areas, even though it is widespread at the seedling stage, and septoria nodorum blotch, which is not controlled by the MBC's is of greater importance.

As can be seen from tables 3 and 5, in all cases where an MBC fungicide was applied to control eyespot, a high degree of control of septoria tritici blotch was achieved. These data were from one trial at the Norfolk Agricultural Station and from six fields sampled on a large commercial estate. That the control was not complete reflects the timing of the MBC application, which, for eyespot control, is later in the season than a spray to obtain complete control of septoria tritici blotch.

SUMMARY

Although there is increasing evidence that both M. graminicola and L. nodorum can colonize wild grasses and hence could act as a reservoir for primary inoculum (Krupinsky, Prestes pers. com.), crop residues must still be considered the most likely source of primary inoculum. Whether this inoculum is in the form of pycnidiospores or ascospores

Table 4.--Percentage yield increases from four year's spray trials to control Septoria tritici blotch. Lincoln, New Zealand. (Early control represents the control of the primary inoculum of the primary inoculum on the wheat seedlings at the 5-leaf stage. Late control represents the control of Septoria tritici blotch on the flag leaf).

	1978-79	1977-78		1976-77		1975-76	
No spray	100	100	100	100	100	100	100
Complete control	120	104	110	167	138	125	-
Early control	112	108	107	150	129	113	106
Late control	115	108	104	143	124	115	104

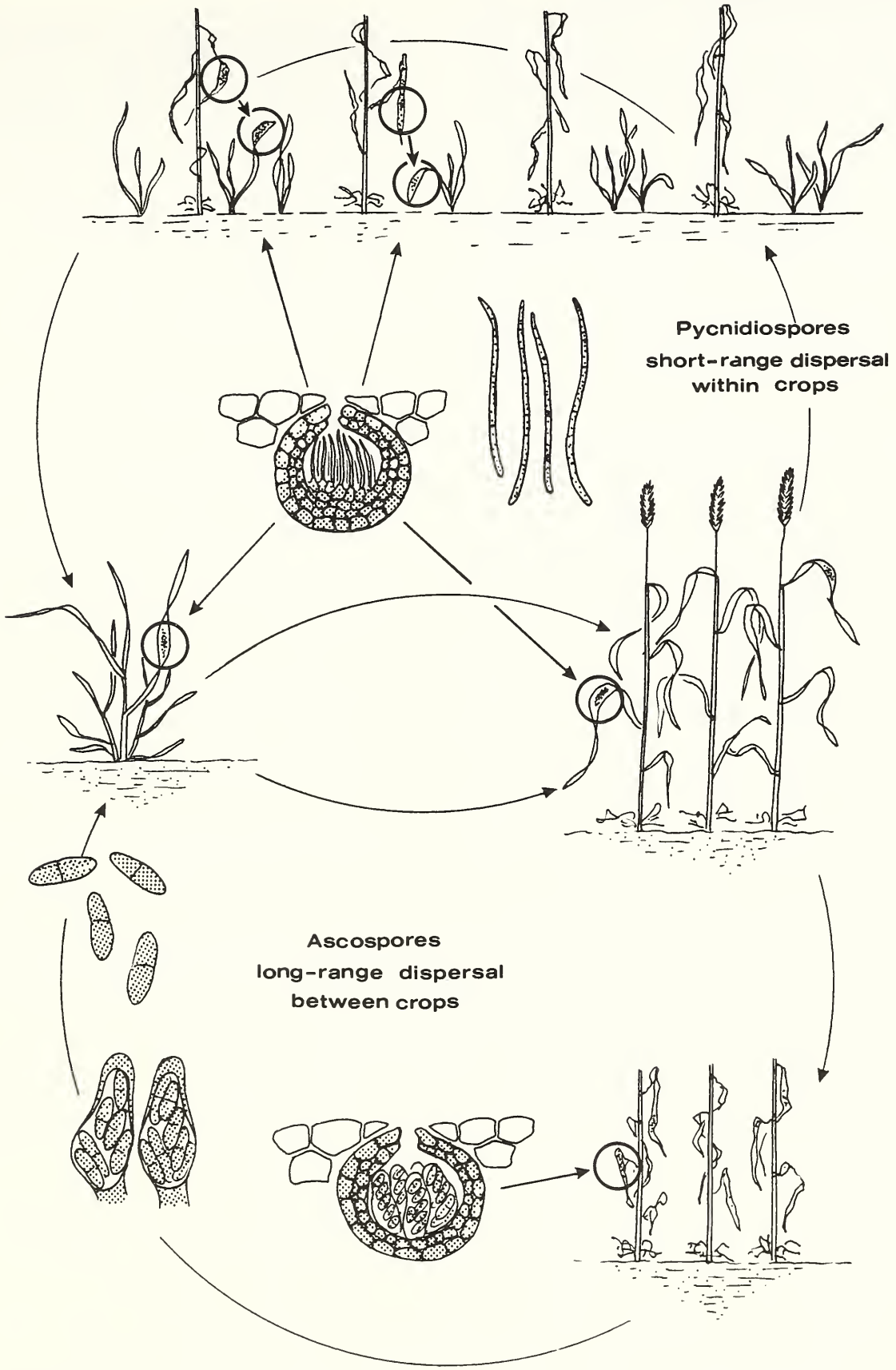
Table 5.--The effect on the inoculum potential of M. graminicola and L. nodorum when wheat crops are sprayed for the control of the eye spot, demonstrating good levels of control of Septoria tritici blotch and poor control of Septoria nodorum blotch.

	Spore numbers	
	M. Gram	L. Nodorum
Norfolk Agricultural Station		
c.v. Flanders sown 16 October		
No benomyl	196	28
+ benomyl	11	11
Morley's		
c.v. Hustler sown 27 September		
No benomyl	161	23
+ MBC	3	7
+ MBC + triadimefon	6	4
c.v. Hobbit		
No benomyl	266	81
Long History Wheat: + MBC. 1/2 triadimefon	50	77
After break + MBC	6	74

depends on the weather condition to which that crop debris is exposed. If conditions are such that there is little or no breakdown of the leaf tissue, and the subsequent crop is sown into crop trash, then the primary inoculum is likely to be rain splash dispersed pycnidiospores (fig. 4).

Where conditions are conducive for the breakdown of leaf material on the soil surface, and where conditions are favorable for pseudothecial development, then the primary inoculum is likely to be ascospores produced on standing stubble, which have the potential to travel long distances to infect crops hundreds of kilometers from the source of origin.

Figure 4.--Diagrammatic representation of the relationships between short-range dispersal of pycnidiospores and long-range dispersal of ascospores of Mycosphaerella graminicola.



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SANDERSON - SPEAKER

Q. Fehrmann: You claim that in England S. tritici is important. How do you justify that?

A. I consider Mycospharella as a seedling disease. Now whether it gets up on the plant depends on conditions late in the season, but there is a seedling disease, I believe, that causes sufficient damage to be considered important.

INCREASE OF INOCULUM OF SEPTORIA NODORUM DURING WINTER

Barry M. Cunfer[1]

Several experiments were conducted to quantify the increase of inoculum of Septoria nodorum during the winter in Georgia. The tests included comparisons of various wheat cultivars, plant density, level of seed infection, cropping history, and chemical seed treatment. In each test, 10 lower leaves were selected randomly from each of four replicated field plots three times during the winter season. Leaves were washed in tapwater, surface sterilized with sodium hypochlorite, and rinsed twice. They were plated on Bannon's medium and incubated at 20°C with 12 hr of light per day. Plots in the first two experiments were 7.4 m^2, and plots in the third test were 1.9 m^2. All plots were separated by borders 1 to 2 m wide.

Experiment 1. Eight wheat cultivars were sampled during two seasons. The number of infection sites increased on all cultivars during the winter. By the March sampling there were significant differences among the cultivars. 'Holley', the most susceptible cultivar, had 2.7 and 4.1 infection sites per leaf in early March in two seasons. The differences in numbers of infection sites among cultivars were in general agreement with their field ratings for susceptibility. Resistance to S. nodorum in early growth stages is the same as that expressed in the adult stage.

Experiment 2. Wheat and barley were planted as pure stands or as mixtures in ratios of 2:1, 1:1, and 1:2. 'Volbar' barley was used in each test. In the first season, 'Stacy' wheat was used; in the second season duplicate experiments were set out, one with Stacy and one with Holley as the wheat component. The number of sites was greater on Holley, which reflected its greater susceptibility. The number of infection sites was lowest when wheat comprised 0.33 of a mixture. There was little difference in site numbers at 0.5 and 0.67 wheat. At the March sampling date, the number of sites was greatest in pure wheat in both tests during the second season but was less than 0.67 wheat in the first year's test. This lower value may have been due to sampling variability and lower number of sites during that season.

The wheat biotype of S. nodorum was consistently recovered from the lower leaves of barley. These leaves can be colonized to a small extent by the wheat biotype when they are green and probably are invaded saprophytically as they begin to senesce. Colonization of barley leaves increased as the proportion of wheat in the mixture increased. The number of sites on barley was greater in the mixture of Holley wheat, which reflected the greater amount of inoculum from this more susceptible cultivar.

Experiment 3. One of Holley wheat grown in a greenhouse (0% seed infection) and a second lot from field-grown seed (48% infection) were planted in duplicate experiments. One test was planted on land that had been in wheat the previous two seasons. The other test was planted on land planted to legumes the previous two seasons. Various seed treatments were applied to the infected seed lot, and there was nontreated controls of each seed lot. Tridimenol and CGA 88531 completely controlled seedling symptoms. Other treatments, including carboxin + thiram, significantly reduced seedling symptoms, but a small percentage of seedlings exhibited symptoms. Neither clean seed nor seed treatments were effective in suppressing infection sites on the lower leaves during the progress of winter on land in wheat the two prior seasons. The number of infection sites was considerably less among the treatments on land not in wheat the two previous years. Significant control continued into March

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Table 1.--Increase in inoculum of Septoria nodorum during the winter on 8 wheat cultivars.

Cultivar	Number of infection sites[1] Mar. 3, 1982	Mar. 7, 1983
Holley	107 abc[2]	163 a
Florida 301	57 bcd	142 abc
Omega 78	39 cd	103 cde
Coker 762	27 cd	73 def
Stacy	22 d	128 bc
Oasis	21 d	46 f
Coker 797	19 d	158 ab
Coker 747	12 d	63 ef

[1]Assays from a total of 40 leaves from 4 replicates.
[2]Values in columns followed by the same letter are not significantly different according to Duncan's new multiple range test (P=0.05).

Table 2.--Number of infection istes of Septoria nodorum in pure stands and mixtures of barley and wheat on March 14, 1983.

Proportion of barley or wheat in mixture		Number of infection sites	
		Volbar-Stacy	Volbar-Holley
1.0	Barley	48 b[1]	57 b
0.67	Barley	74 ab	113 a
0.50	Barley	107 ab	131 a
0.33	Barley	148 a	153 a
1.0	Wheat	172 a	223 a
0.67	Wheat	116 a	184 ab
0.50	Wheat	115 a	180 ab
0.33	Wheat	97 a	149 b

[1]Values in columns within each cereal species followed by the same letter are not significantly different according to Duncan's new multiple range test (P=0.05).

Table 3.--Infection of Holley wheat by Septoria nodorum during the winter in fields with and without the two previous years.

Treatment	Percent seed infection	Percent seedlings with coleoptile lesions[2]	Number of Infection sites[1]					
			Land in wheat 2 yr			Land not in wheat 2 yr		
			Jan. 6	Feb. 8	Mar. 7	Jan. 6	Feb. 8	Mar. 7
Control not sprayed.	0	3.5	113	156	124	1	0	43
Chlorothanlonil (foliar) 2 pt/A.	0	3.5	69	82	112	0	0	20
Control	48	44	64	91	134	21	70	192
Tridimenol (seed) 2.2 g/kg.	48	0	65	99	164	3	2	77
Carboxin + thiram (seed) 2.5 g/kg.	48	6	108	133	144	11	15	58
CGA 88531 (seed) 0.8 g ai/kg.	48	0.3	--	--	--	--	--	16

[1]Assays from a total of 40 leaves from four replicates per treatment at each sampling date.

[2]Four replicates of 100 seedlings harvested one month after planting.

in plots planted with clean seed and tridimenol and CGA 88531 seed treatments; however, secondary spread from control plots and the less effective treatments was evident by then. Control of seedborne inoculum is effective only if used in combination with crop rotation.

Septoria nodorum is capable of increasing its inoculum throughout the winter in the southeastern United States. Sporulation and secondary spread of inoculum are also evident.

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CUNFER - SPEAKER

Q. Spadafora: How did you determine whether the inoculum was seedborne or airborne?

A. A student at North Carolina State had some data a couple years ago. In the abstract, it states that they did find up to a 20% increase with seed infection. I've tried a number of times to differentiate inoculum sources, but whether its's pycnidiospores or ascospores, I don't know. You've got to take a lot of land and have it isolated.

Q. Do you have any problems of contamination with your plots being too close together?

A. Not with rotation. I have plots but not far enough apart. Herb Luke and Ron Barnett in Florida have done some of these experiments, and they have found some decrease in disease associated with rotation and tillage practices.

Q. Fehrmann: How long does your stubble remain a source of inoculum?

A. Under our conditions, where we have mild seasons, our stubble does decompose more rapidly. This is the thing that I'm concerned about because almost everybody else is working in winter climates whereas I'm working in a moderate climate.

Q. Prestes: As most of you know, we grow wheat in winter and soy beans in summer in the same field. I have found late in autumn that a lot of septoria remains in wheat stubble in soy bean fields. You mentioned rotation as a means of reducing septoria. Do you think that once you go to no tillage in the summer, you increase a lot of septoria?

A. Our climate is very similar to yours. In fact, that's the way our wheat is grown; soy bean planted in wheat stubble. I have not been able to document that there is an increase of S. nodorum because of stubble on the surface. I didn't really want to emphasize seed inoculum, but my main point was to identify the source. If inoculum is there from the time the seedling germinates, I think that nodorum can increase during the winter seasons.

Q. Scharen: I was wondering if Dr. Sanderson would like to comment on increase of inoculum during the winter and what you found in standing stubble from our plots which were inoculated last year.

Comment by Sanderson: What goes through on the stubble the next year is going to depend entirely on weather conditions they are subjected to. In Bozeman, during the winter, the stubble supports the pycnidia of both nodorum and tritici. The organisms have gone through in weather conditions such that you get production of viable pycnidia. We have found L. nodorum but not Mycosphaerella. From what we saw in central Oregon, stubble goes through the winter and comes out with nothing. In New Zealand, we go through the winter and get an abundance of ascospores, so you've really got a lot of variation in the weather conditions that stubble is subjected to. Variation must be considered before you can find out what the primary inoculum source is going to be.

PRELIMINARY STUDIES ON THE EFFECT OF INTERRUPTED WET PERIODS ON INFECTION OF WHEAT BY SEPTORIA NODORUM

J. Robert Tomerlin[1]

The establishment of infection by Septoria nodorum is strongly dependent on environmental factors, particularly the availability of moisture. Septoria nodorum may incite infection when the foliage is wet or when subjected to high relative humidity. Different wetting periods are required on different wheat cultivars, with 2 hr being adequate to initiate infection in very susceptible spring wheat cultivars (1), although postinoculation wet periods of 72 to 96 hr are considered optimal. Eyal et al. found excellent correlations between length of postinoculation wet period and area of necrosis and number of lesions (2).

Although most workers generally subject inoculated seedlings to continuous moist treatments, protracted conditions of continuous moisture do not often occur in the field. This study was conducted to investigate the influence on disease development of postinoculation wet periods comprised of either high relative humidity or leaf moisture, the interruption of the wet period with a period of dryness, and high versus low relative humidity after infection was established.

A virulent isolate of S. nodorum was maintained on Czapek-Dox V8 juice agar under continuous light at 20°C to induce sporulation. A spore suspension of 2×10^6 spores/ml was prepared from 8-day-old cultures. One drop of Tween 20 was added per 100 ml of inoculum. Twelve-day-old seedlings of Fortuna wheat were sprayed with a light oil and inoculated with the spore suspension. After inoculation, seedlings were either placed in mist chambers resulting in leaf moisture or in a high humidity chamber (RH > 96%) in which foliage remained dry. Dry periods during the postinoculation wet period were imposed in the following patterns: 72 hr of moisture were interrupted by 8 hr of low relative humidity; 0, 8, 16, 24, 32, 40 or 48 hr after inoculation; 24 hr of low relative humidity 24 hr after inoculation; or 8 hr of low relative humidity every 16 hr. In addition, some plants were subjected to wet periods of 72, 80, 96, or 104 hr without dry interruptions. After the establishment of infection, seedlings were maintained at either high or low relative humidity (RH < 61%).

Disease severity was evaluated using the Horsfall-Barratt scale (3), and values were converted to percent leaf area diseased. Dry period treatments were evaluated using single degree of freedom contrasts.

The experiment was intended to allow comparison of moisture source (leaf wetness or high relative humidity) during incubation. The high humidity treatment was provided by a humidifier in a plastic enclosure in the greenhouse, in which extremely high temperatures occurred. Therefore, the high humidity treatment will not be discussed in detail because of confounding with high temperature. Temperature in the high and low humidity postinfection treatments were similar, allowing comparison between high and low humidity after inoculation.

Plants maintained at low relative humidity after inoculation had less disease than plants maintained at high relative humidity. Regression of percent leaf area infected on the time of delay of the dry interruption (not including the 0 hr delay) was significant. Regression of percent diseased area on length of wetting of 72 hr and greater was not significant. A dry period of 8 hr every 16 hr resulted in lower disease levels than continuous wetting (table 1).

Although the interaction of dry treatment and postinoculation humidity was not significant, percent leaf area diseased was greater for the 0 hr delay treatment than for the 8 hr delay treatment.

The effect of moisture on development of S. nodorum is complex. These preliminary studies indicate that after infection is established, disease develops to a greater extent under high relative humidity than under low relative humidity. A dry interruption of the wet period occurring within 24 hr of the application of spores may result in less disease development. These findings are in general agreement with those of Jeger et al. (4), who observed decreased infection in the field when 4 hr of relative humidity of less than 90% occurred in the first 24 hr following inoculation.

The effect of intermittent drying may result from a requirement for moisture by various stages of the infection process. For example, drying may be deleterious if a spore germinates and is subjected to dry conditions before penetration. Further experiments are planned to investigate the role of moisture in establishing infection of S. nodorum.

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Table 1.--Effect of moisture treatment on disease development on seedlings of Fortuna wheat inoculated with Septoria nodorum.

Treatment	Relative humidity	
	High[a]	Low
0 hr delay[b]	69[c]	65
8 hr delay[b]	65	17
16 hr delay[b]	74	21
24 hr delay[b]	90	46
32 hr delay[b]	93	24
40 hr delay[b]	83	28
48 hr delay[b]	94	69
72 hr wet	90	83
80 hr wet	93	60
96 hr wet	90	77
104 hr wet	77	60
24 hr dry[d]	88	21
Alternate dry[e]	65	24

[a]Plants were maintained in either high or low relative humidity after infection was established.
[b]Plants subjected to 72 hr of moisture, interrupted by 8 hr of low relative humidity n hr after inoculation.
[c]Value is percent leaf area infected following conversion from Horsfall-Barratt value.
[d]72-hr wet period with a 24-hr dry period 24 hr after inoculation.
[e]72-hr wet period with an 8-hr dry period every 16 hr.

EFFECT OF MOIST PERIOD DURATION ON SEPTORIA TRITICI BLOTCH OF WHEAT

Dale E. Hess and Gregory Shaner[1]

Little specific information exists on the relation of weather to epidemics of Septoria tritici blotch of wheat (caused by Mycosphaerella graminicola (Fuckel) Schroeter) although early studies suggest that abundant rain with foggy or cloudy conditions favors the disease (3, 8). Moisture is known to be important at all stages of the infection cycle, but it is unclear how much moisture, and for how long, is needed in the field for an epidemic to occur. Fifteen hours of leaf wetness has been reported to be a minimum for infection in the field (5). In the lab, spores germinated on wet leaves within 12 hours and penetrated after 24 hours (1). Satisfactory infection is obtained in greenhouses by keeping inoculated plants in a moist chamber for 2 to 4 days (2, 4).

In our experience, climatic conditions not conducive to natural infection and disease spread result in poor disease development regardless of the frequency and timing of inoculations. In the field, extended moisture periods may be more important than artificial inoculations for increasing disease severity. Septoria tritici blotch is only a problem in abnormally wet years (6, 7).

Experiments were conducted in the greenhouse to investigate the effect of different post-inoculation moist periods on disease severity at different temperatures.

MATERIALS AND METHODS

Four cultivars of soft red winter wheat (Triticum aestivum L. em. Thell) were employed: highly susceptible Morocco, moderately susceptible Beau and Arthur, and resistant Auburn.

Conidia of M. graminicola were produced in abundance on the surface of yeast malt agar. Inoculum was prepared by scraping spores from the agar surface and diluting them in water to a concentration of 10^6 spores per milliliter. Unflavored gelatin (0.5g dissolved in 25ml of warm distilled water) was added to 100ml of inoculum as a sticker.

Plants were arranged in a randomized block design on a greenhouse bench, and a DeVilbiss atomizer 151 was used to inoculate plants on the adaxial surface of the flag leaf. A density of approximately 46 spores per mm^2 was obtained. Plants were at the seed-filling growth stage.

Following inoculation, plants were placed into a moist chamber and kept moist by a time clock-controlled misting system. Fifteen plants of each cultivar were removed following 24, 48, 72 and 96 hours of moisture and five were placed into each of three growth chambers preset to maintain temperatures of 11, 18, or 25C. The experiment was performed twice.

An attempt was made to simulate cold nights following natural infection of wheat in the spring. Cultivar Morocco was used and replicated six times. Plants were at the seed-filling growth stage and exposed to a 96-hour post-inoculation moist period, during which there were various intervals of low temperature (table 1). To maintain surface moisture during exposure to cold in a 5C coldroom, plants were atomized with distilled water and covered with polyethylene bags. The mean temperature within the moist chamber was 27C.

Following the 96-hour moist period, plants were removed to a 28C growth chamber. Disease severities were recorded daily from 14 to 26 days post-inoculation.

RESULTS AND DISCUSSION

Results for moist period duration experiments are an average of two experiments. Data for Auburn in the 25C growth chamber were omitted, as results for only one experiment were available.

First, post-inoculation moist periods were compared for a given cultivar and temperature regime (table 2). Similar levels of disease resulted from 72 and 96 hours of moisture while 48 hours produced significantly less disease. A moist period of only 24 hours was generally insufficient to produce disease symptoms. Cultivar Morocco at 25C was a notable exception.

Temperature regimes were compared for a given cultivar and moist period (table 3). Within a given moist period, higher temperatures resulted in higher disease levels.

The reactions of different cultivars within a given moist period and temperature regime (table 4) were also examined. Cultivar Auburn maintained low disease levels for all temperatures and moist periods. Cultivars Beau and Arthur reacted similarly, exhibiting intermediate levels of disease. Cultivar Morocco was consistently most susceptible.

Densities of pycnidia produced within lesions on the different cultivars increased with increasing temperature (fig. 1). Cultivar Auburn produced fewest pycnidia within lesions but the differences seen between the highly and moderately susceptible cultivars with respect to lesion development were not maintained.

Results of the coldroom experiment are shown in figure 2. Disease curves were similar for all treatments but increasing exposure to cold resulted in a corresponding decrease in disease severity. Curves for 30 and 34 hours of cold were identical.

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Table 1.--Treatments in cold night simulation experiments.

0 hours cold = control
30 hours cold = first night (10 hr) in moist chamber, 3 subsequent nights in coldroom
34 hours cold = first 2 nights and 1 intervening day in coldroom
40 hours cold = first 4 nights in coldroom
58 hours cold = first 3 nights and 2 intervening days in coldroom

Table 2.--Severity of Septoria leaf blotch on the flag leaf of four wheat cultivars[a] [b]

Cultivar and growth chamber temperature, C (average of 2 experiments).

	Morocco			Beau			Arthur			Auburn		
Post-inoculation moisture (hours)	11	18	25	11	18	25	11	18	25	11	18	25
24	8c	14d	42b	3c	8b	11c	4c	7b	9d	3b	3b	--
48	27b	44c	73a	11c	21b	29b	10c	16b	33c	3b	5ab	--
72	57a	60b	82a	38b	48a	72a	33b	45a	56b	6b	9a	--
96	66a	76a	84a	52a	52a	77a	56a	51a	74a	20a	10a	--

Table 3.--Severity of Septoria leaf blotch on the flag leaf of four wheat cultivars[a] [b]

Cultivar and post-inoculation moisture period in hours (average of 2 experiments).

	Morocco				Beau				Arthur				Auburn			
Temperature (C) of growth chamber	24	48	72	96	24	48	72	96	24	48	72	96	24	48	72	96
11	3b	5c	19c	30c	3c	3b	8c	13c	3c	3c	4b	12c	3a	3a	3a	3a
18	14b	44b	60b	76b	8b	21a	48b	52b	7b	16b	45a	51b	5a	3a	9a	10a
25	42a	73a	82a	84a	11a	29a	72a	77a	9a	33a	56a	74a	--	--	--	--

Table 4.--Severity of Septoria leaf blotch on the flag leaf of four wheat cultivars[a] [b]

Post-inoculation moisture period in hours, growth chamber temp., C (Av. of 2 experiments).

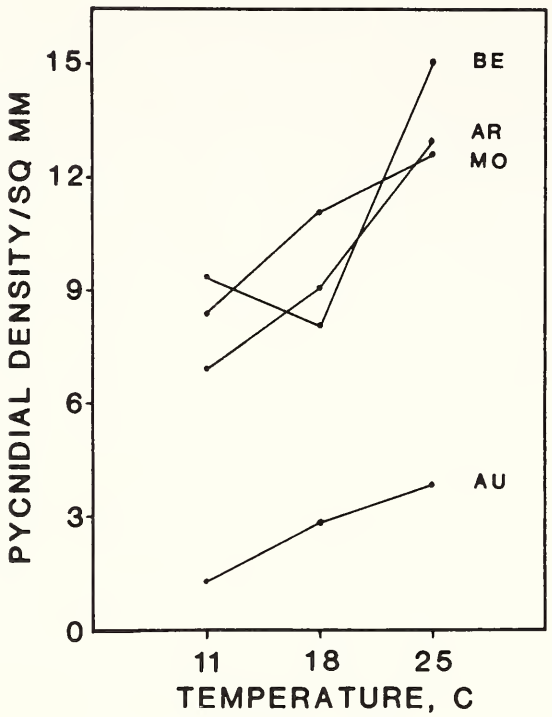
	24			48			72			76		
Cultivar	11	18	25	11	18	25	11	18	25	11	18	25
Morocco	8a	14a	42a	27a	44a	73a	57a	60a	82a	66a	76a	84a
Beau	3b	8b	11b	11b	21b	33b	38b	48a	72a	56ab	52b	77a
Arthur	4b	7b	9b	10b	16b	29b	33b	45a	56b	52b	51b	74a
Auburn	3b	5b	--	3c	3c	--	6c	9b	--	20c	10c	--

[a]Within each column, means followed by a letter in common are not significantly different (Duncan's new multiple range test, $\alpha = 0.05$).

[b]Columns show mean arcsin - square root severity. A value of 3 corresponds to 0% and a value of 87 corresponds to 100% necrotic leaf area because 0 and 100% were converted to to 0.25 and 99.75%, respectivel, before transformation.

Note: In tables 2 and 4, readings for the 11, 18, and 25C growth chambers are from 32, 26, and 26 days post inoculation, respectively. In table 3, all readings are from 26 days post inoculation. The footnotes in table 4 apply to all three tables.

Figure 1.--Densities of pycnidia of Mycosphaerella graminicola within lesions on four cultivars of wheat at three temperatures. BE = Beau, AR = Arthur, MO = Morocco, and AU = Auburn).



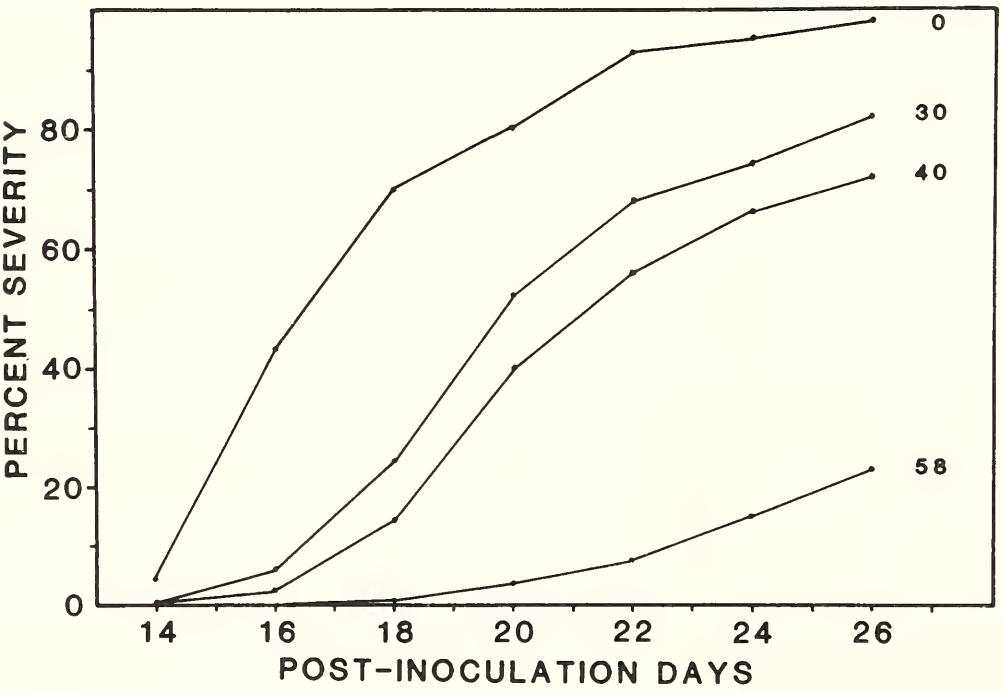
CONCLUSIONS

It appears that there is a compensation effect between moisture and temperature in susceptible wheats. Where the moist period is short, high temperature may still produce severe levels of disease. With long moist periods and low temperatures, again high disease levels are observed. In resistant wheats disease severities are low under all conditions.

Pycnidial densities within lesions increase with increasing temperatures but do not necessarily correspond to the relative susceptibilities, in terms of blotch severity, of wheats. It will be worthwhile to examine the numbers of spores produced within the pycnidia.

Further investigation must be made of the effect of cold on disease development. It is unclear whether spores were killed or merely inactivated by cold temperatures. In the latter case an extended moisture period at favorable temperatures may reactivate them.

Figure 2.--Disease progress curves for Morocco wheat exposed to four different post-inoculation cold treatments. (0, 30, 40, and 58 = total hours at 5C during a 96- hour moist period).



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HESS - SPEAKER

Q. Hosford: Something that is a general question is why Septoria tritici requires such a long wet period. There is a group of minor pathogens that require a long wet period for infection. They are not serious at all. Why is Septoria tritici such a major pathogen but yet requires such a long wet period. I don't think we have the answer to it.

A. I am in no position to give an answer to that. It's a very interesting question.

Q. Eyal: I think that we are measuring two different effects here, but I think that Ricardo Madariaga can tell us a little bit what he has done with temperature regimes and moisture periods.

Comment by Madariaga: To study latent period, I used four types of temperature regimes, moisture was standard. I cannot say just temperature regimes since temperature was not constant. The term "environment" seems more appropriate. Temperatures changed in all chambers each 12 hours. In some of the growth chambers, changes were more gradual. We found that at 12 hours at 5°C, with no light and 12 hours at 10°C with light, the latent period was almost 30 days with differences between genotypes. The variety Anza showed pycnidia formation only in this environment. The other extreme was a warm environment of 5°C, with no light, and 32°, with light. Symptoms did appear, but pycnidia formation was not detected. There is an interaction between genotype and temperature, so it is necessary to express a specific latent period.

Q. Krupinsky: Did you evaluate the lower leaves?

A. Hess: I didn't because, when working with only the lower leaves, there is difficulty with natural senescence, and it is more difficult to decide if this result is disease or not. For simplicities sake, I just worked with the flag leaf.

GLOBAL "FINGERPRINTING" of LEPTOSPHAERIA NODORUM (SEPTORIA NODORUM) AND MYCOSPHAERELLA GRAMINICOLA (SEPTORIA TRITICI) PATHOGENICITY PATTERNS

Z. Eyal, A. L. Scharen, and J. M. Prescott[1]

Leptosphaeria nodorum E. Müller, the causal agent of the septoria nodorum blotch of wheat, and Mycosphaerella graminicola (Fuckel) Schroeter, the causal agent of septoria tritici blotch of wheat, cause considerable damage to the crop in several parts of the world (1, 4, 6). In many of the studies concerning these pathogens mixtures of isolates from diverse sources have been utilized to ensure a representative virulence spectrum (6, 7). Some authors have referred to the presence or absence of physiological races or differential host-pathogen interactions among the obviously different populations of the pathogens (1, 6, 7). In most of these, substantial cultivar-isolate interactions were reported (6). Quantitative inoculation techniques introduced a higher degree of accuracy and repeatability, based on quantitative host response assessment (2). Some wheat cultivars reported to be resistant in one country did not express the same resistance levels when subjected to septoria populations and environmental conditions in another country (1, 6, 7). Moreover, some wheat cultivars resistant to certain populations of the pathogen in one location within a country have expressed high disease severity to populations or single isolates secured from different locations (6, 7). The magnitudes of cultivar x isolate interactions within a location, country, or geographical region have not been explored, though these interactions may have important roles in utilizing germplasm, deploying varieties, planning breeding programs, and designing pest management strategies.

The realization of these ideas was carried out in the present study. Isolates of both pathogens were secured from leaf samples originating from eight countries in the L. nodorum portion of the study and 22 countries in the M. graminicola part. Thirty three L. nodorum isolates and 97 M. graminicola isolates were quantitively inoculated at the seedling stage on a set of 50 wheat (spring and winter bread, and durum) and triticale cultivars, using adjusted spore concentrations, and uniform inoculation, incubation, and assessment procedures (2, 6). The set of cultivars was assembled for both pathogens, taking into consideration host response (resistant and susceptible) as previously reported (5, 6, 7). Obvious limitations in sampling of the pathogen within a country, selection of suitable cultivars, and nonuniform laboratory analytical procedures categorizes the study as a "fingerprinting" or as an insight into pathogenicity patterns, rather than a complete pathogenicity survey.

When present, host response was assessed on the basis of percent of necrotic leaf area and the percent coverage by pycnidia (M. graminicola). The uniformity and repeatability of methods were ascertained by the inclusion of standard check cultivars and isolates in each test over the span of trials. The variability among plants within an experiment was rather small and not significant, and the interaction between check cultivar x check isolate x experiments was relatively low.

Analysis of an isolate x cultivar matrix formed from an "all possible subsets" regression model or a two-way analysis of variance were used to estimate predicted means and standard errors. The "dividing line" between a susceptible and a resistant host response was estimated from a cluster analysis over the entire isolate x cultivar x plant matrix, where the confidence interval for the calculated dividing line was derived using the overall residual mean square. The dividing line for the L. nodorum matrix was calculated to be 17.94% necrosis and 16.57% necrosis for the M. graminicola study.

Pathogenicity patterns were estimated using DIFFER and GENEALOGY computer programs (3), which estimate the minimum number of genes assuming a gene-for-gene relationship. Due to the size of the analyzed matrices (33 isolates by 50 cultivars for L. nodorum and 97 isolates x 50 cultivars for M. graminicola) no attempt was made to assign specific designations to the hypothesized genes. Rather, the analysis was used to calculate overall virulence frequencies or specific virulences in six geographic regions and in certain countries within each region. As mentioned previously, the number of tested isolates per country or region varied markedly, but important conclusions can still be drawn especially for M. graminicola. The regions and countries varied considerably in the relative virulence frequencies with South America having the highest virulence level (fig. 1). Uruguay and Mexico had the most virulent populations of the pathogen while Chile had the least virulent. In certain countries, virulence was rather restricted in its spectrum being specific to certain host genes or to wheat classes (durum-derived lines from Russian bread winter wheat or Frontana-derived South American lines).

Some resistant wheat cultivars were rather effective throughout the virulence spectrum while some very susceptible wheats expressed a resistant response to certain specific isolates. A knowledge of host response to specific biotypes within a population or to populations may thus aid in selecting proper and diverse germplasm, identifying the resistance sources effective, and, consequently, designing the proper breeding strategies in many national and international research programs. This knowledge may further suggest deployment strategies for germplasm and later for cultivars according to pathogenicity patterns.

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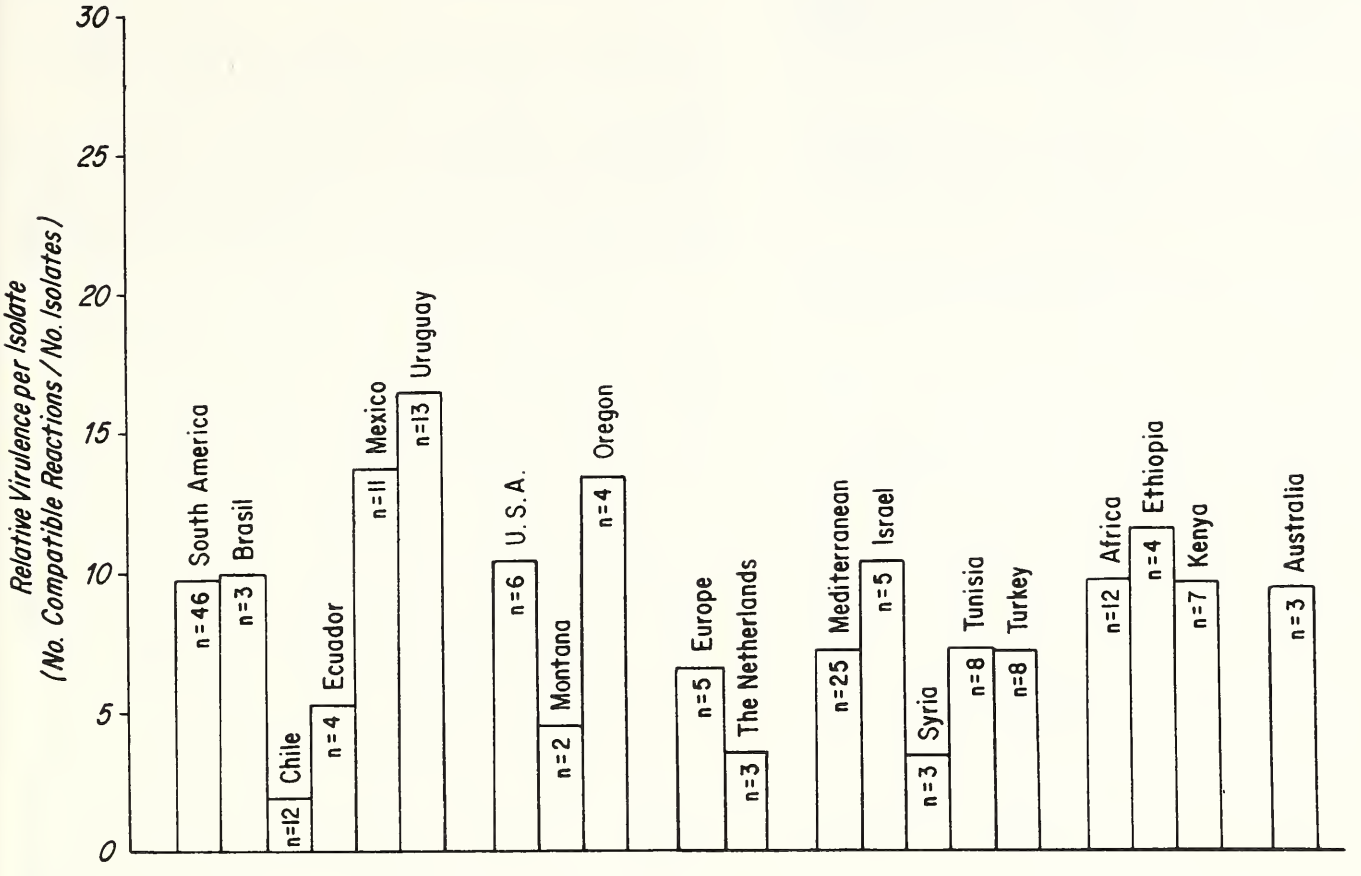
Figure 1.--Relative virulence os Septoria tritici in several areas of the world.



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Q. Bob Hosford: It is interesting that Olaf was developed in North Dakota where we have very high virulence of Septoria tritici on Olaf and have no S. tritici.

A. Prescott: We have had similar situations in Mexico where Siete Cerros is used as a susceptible check, but it was not developed in an area where Septoria tritici is a problem.

Q. Peter Scott: I am very interested in possible pathogenicity variation and variation in varietal response.

A. Prescott: Yes, I think there is variation in pathogenicity and also in varietal response. There is clear-cut evidence. In Turkey, isolates and disease data from the western coast, which is isolated from the southern coast, give very clear-cut differences.

Q. Scott: In your experiment, for example, was there any evidence of interaction between isolates and cultivars? Did you present that data?

A. Eyal: Everywhere we find an interaction between host and parasite we will say we have physiological specialization. In a recent paper from North Carolina, another factor, namely a host by pathogen by experiment interaction, was postulated, and all of the interactions were quite high. When we derived that experiment, the main effect was the host by isolate interaction, which was quite high. The frequency of the virulences here were assigned from the hypothesized particular virulent gene, and we prefer to refer to these genes by the cultivar on which they were virulent.

Q. Scott: I have a related question. Was it clear from the beginning that there was a specific host range?

A. Eyal: In any physiologic specialization work in which you analzye host by parsite interaction, you cannot be 100% sure of plant responses. Therefore, you have to decide where you are cutting resistance and susceptibility. We have decided, for this particular experiment, to set up a statistical procedure in which we recommend they submit information and we analyze it. We have decided on this matrix to measure the components.

Q. Scott: As soon as you make a statement like that, you are making an assumption that there is a gene for gene relationship.

A. Eyal: Why shouldn't we assume that there is a gene for gene relationship? If you have such an interaction, you have to give me a very strong argument otherwise. We can accept or not accept what Ellingoboe said, but let's say that he is right, so we will follow his assumption that in every host parasite interaction, this system is operating.

Q. Peter Scott: I'm not going to say that they're wrong, of course.

A. Eyal: You can analyze gene frequency many other ways. The best way for us, based on this assumption, is to use this system, and it works quite well. We can predict what happened, for example, in Oregon. Dr. Prescott can verify why in Oregon, Kavkaz broke down when they used it extensively because they never realized that they had the Kavkaz virulence. Worldwide in the Middle East, too, you can pick up so much virulence on the durums.

Q. Caten: In your analysis of variance on your scores of percent necrosis, have you any idea what the proportion is of variation that comes out in the host by pathogen interaction?

A. Eyal: The main effects are very, very large. The interaction, in many cases, is quite large. The values for the interaction will be in the 20's or the 30's; the main effects were smaller.
Q. Caten: So it is quite a bit smaller. If you have got the true gene for gene system with extreme physiological specialization, then the interaction is as big as the main effect in the extreme gene for gene types.

A. Eyal: Not necessarily. You can look at some of the data and you can see that there is quite a variation in the mean square of the interaction.

Q. Peter Scott: In your plot of Septoria nodorum and Septoria tritici, you don't have quite as high a positive correlation. I assumed that the varieties which are susceptible to nodorum are also susceptible to tritici.

A. Eyal: That is what the high correlation implies.

Q. Scott: I was surprised to see that high a correlation at all.

A. Eyal: A study of this has determined that the source of variety is important. Dr. Scharen and I decided to pick up varieties that were resistant and that we had selected throughout the years. We knew that, to some extent, they had resistance to one organism, in many cases to both. So I think the source of resistance of course effects the study.

Q. Caten: I think you said the second part of your program provided an estimate of the number of resistance genes to nodorum and tritici.

A. Eyal: In the 97 by 15 matrix, results from the analysis estimated there were 27 corresponding genes. When we did the same program and a 47 by 7, we estimated nine genes for resistance to nodorum. We are attempting to verify the hypothesized number of genes with actual number of genes that we figured out from the actual study. Therefore, we are not quoting more than just a hypothesis.

REDUCED GREEN LEAF AREA AND YIELD LOSS CAUSED BY SEPTORIA TRITICI

R. E. Gaunt[1]

Field trials conducted over several seasons indicated that the effect of speckled leaf blotch (Mycosphaerella graminicola (Fuckel) Schroeter, imperfect state Septoria tritici Rob. ex Desm.) on yield was due to reduction in grain number per ear and grain weight, with smaller effects on ear number per plant (3, 4, 10). The results suggest that disease prior to flowering was at least as important as that during grain filling.

In the 1979/80 season we investigated in detail the effects of speckled leaf blotch on plant growth and development to gain an understanding of the effect of disease on yield potential and development.

As a preliminary we investigated methods of disease assessment (5) in view of the work (6, 7) on disease assessment in barley. In the barley work we concluded that a method of disease assessment based on host parameters was more likely to be useful for yield loss studies than a method based on pathogen parameters. This conclusion has been supported by Carver & Griffiths (1, 2).

Disease was measured in a four-replicate randomized block field trial, at weekly intervals, in each of four treatments: early, late, full and nil disease epidemics. Severity was assessed as the percentage of non-green leaf area on all leaves. Leaf area was also measured at each leaf position. When expressed as a Disease Index (sum of percentage non-green leaf area on top four leaves of diseased plants minus sum for healthy plants) there were two peaks of disease, during September and from late October onwards (fig. 1). When expressed as absolute green leaf area per shoot, disease, measured as the differences in this parameter between healthy and infected plants, had an effect on the plants from late August through to harvest. We concluded (5) that the apparent decline in disease, indicated by the Disease Index, during October was associated with rapid plant growth at this time (Spring) and that the index took no account of the effect of the initial epidemic on leaf area expansion nor on maximum leaf size. We argued that absolute green leaf area was likely to be more closely related to yield potential and final yield than the Disease Index.

Yield was reduced most by the full disease epidemic, with the early phase of the epidemic having a greater individual effect than the late phase (table 1). The number of ears per unit area was not affected in this season by disease. The early disease epidemic reduced the number of grains per ear whereas the late epidemic had no significant effect on grain number nor grain weight. The full disease treatment reduced both yield components significantly. In the early disease treatment there was evidence for compensation for the reduced grain number by an increased grain weight, though this was insufficient with barley (7) we have not observed such compensation and believe that compensation in this season may have been associated with the abnormally wet summer.

When analyzed in detail (table 2) the effect of early disease on grain number was correlated with a reduced number of floret primordia produced, especially on tiller ears, and to reduced grain set in fertile florets. There was no significant effect on spikelet number. In plants exposed to the late epidemic only, there was an effect on grain set in tiller ears only. One can hypothesize that it is important to protect plants from infection during these early stages of apical development,

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Figure 1.--Disease Index and Absolute Green Leaf Area per shoot on winter wheat.

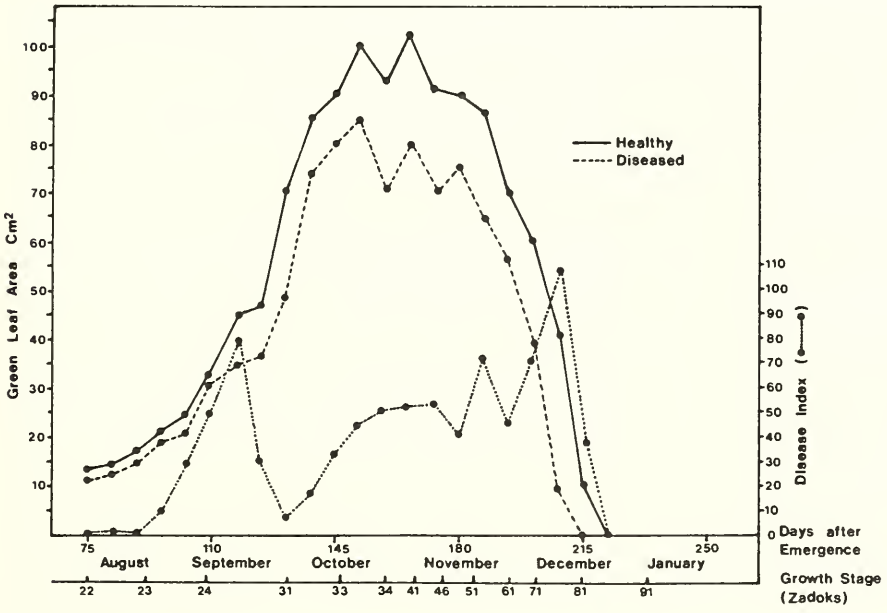

Table 1.--Effect of Speckled Leaf Blotch on Yield components in winter wheat.

Treatment	Yield $g\ m^{-2}$	Ears/m^2	Grain/Ear	Grain Wt mg
Nil Disease	734 d	648 a	35.8 b	31.7 b
Late Disease	726 c	644 a	35.4 b	31.8 b
Early Disease	705 b	638 a	33.4 a	33.1 c
Full Disease	636 a	641 a	34.1 a	29.1 a

Letters denote differences in vertical colums by Duncan's Multiple Range Test ($P < 0.05$).

Table 2.--Effect os Speckled Leaf Blotch on grain number potential in winter wheat.

Treatment		Spikelet No. per ear	Floret Primordium No. per Spikelet	Grain No. per Spikelet
Nil Disease	MS	21.68	8.21 a	2.19 a
	T2	20.24	7.55 cd	1.81 de
Late Disease	MS	21.20	8.21 a	2.08 ab
	T2	19.91	7.55 cd	1.72 f
Early Disease	MS	21.40	8.02 ab	2.03 bc
	T2	20.14	7.21 e	1.64 f
Full Disease	MS	21.49	8.02 ab	1.96 c
	TS	20.12	7.21 e	1.66 ef

T2 is the primary tiller in the axil of leaf 2. Letters denote significance of difference in vertical colours ($P < 0.05$).

though the cause of such reductions has not been elucidated.

When comparing effects on yield potential and levels of disease, it is clear that some reductions in yield potential occur prior to anthesis and prior to the peak of the epidemic. Floret primordium development occurred during the apparent decline of disease in October. This emphasizes the need for an assessment method which adequately describes the effect of disease on plant growth potential.

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GAUNT - SPEAKER

Q. Fehrmann: How did you determine the green leaf area?

A. We assessed the amount of green leaf area using diagrams and subtracting the total leaf area. It's very tedious, and we did that for individual leaves. I think we're fortunate that we're about to break through into technology where we will be able to have that done automatically with the help of a microcomputer.

Q. Jim Frank: If you're using green leaf area, when rating the leaves and looking at rust or powdery mildew you can very easily discern the area covered by the organism. When you're looking at total green leaf area, how do you differentiate diseased area from senesced area or stressed area?

A. We are not concerned so much with the cause of the reduction in green leaf area, but just that it is reduced. This is measurable but not necessarily entirely measurable. Area has been lost because you've got leaves with natural senescence. In long term epidemics, you've got smaller leaves late in the crop growth cycle because of the early effects of the pathogen. All those things we're not too concerned with. We should make straight mention of something we think is related to production. That is a very simple physiological assumption that the pathogen not only reduces photosynthetic area but uses carbon produced by the remaining green leaf area. We are looking at light reflectance as a method of disease assessment.

Q. Nelson: We've had disease epidemics in Texas that were built up very early preanthesis, and we got unusually dry weather and low humidity, which prevented an expected high yield. Do you think this would be a situation that would follow with your results?

A. One can't generalize your production system, and time can effect the disease. You have to look at the yield potential because that is going to determine the relative balance between the production aspect of the plant system and the carbon utilization demand in that system, and that, we think, is one of the main factors that determine whether other factors affect your production system. I think you have to go into your own production systems, test it out and see what happens. I'm not saying the disease before anthesis is more important than after anthesis, but I don't think your data contradict the philosophy. The philosophy is to find out what is constraining your system at specific growth stages. If you know that, that may be the time when disease will have its major effect. We have gone as far, virtually, as saying we will consider diseases simply as one of the constraints along with water, radiation, etc. In the modeling part of our interest, we are trying to build, with physiologists, crop loss models for disease with all the others such as water, radiation, etc.

Q. Masaad: If we compare dry matter of the affected area to the total dry matter in the leaves, it will be a good measure of resistance, and it would be a good score for disease or is it?

A. We have done some work on that and our experience so far says that it doesn't tell us very much about it.

THE RELATIONSHIP BETWEEN SEPTORIA LEAF BLOTCH AND POWDERY MILDEW ON SOFT RED WINTER WHEAT IN PENNSYLVANIA

J. A. Frank[1]

Powdery mildew and Septoria leaf blotch are the two major foliar diseases on winter wheat in Pennsylvania. *Erysiphe graminis* D.C. f. sp. *tritici* E. Marchal is the causal agent of powdery mildew while two fungal species, *Septoria nodorum* Berk. and *Septoria tritici* Rob. ex Desm., are responsible for Septoria leaf blotch. Since symptoms produced by these two *Septoria* spp. are similar and positive differentiation is dependent upon microscopic examination of the conidia, no attempt will be made to distinguish between the species in this paper. They will henceforth be referred to as *Septoria* spp.

Both powdery mildew and Septoria leaf blotch can be found on the same wheat plant, often on the same leaf. These pathogens have been shown to have specific relationships to each other and to other pathogens. Chester (2) demonstrated that infection by *S. tritici* on winter wheat reduced the incidence of leaf rust. Sanderson (4) reported that several pathogens, including viruses, predisposed wheat to *S. tritici*. Brokenshire (1) found that *E. graminis* predisposed a Septoria resistant cultivar of wheat to *S. tritici* infection so that sporulation of *S. tritici* was significantly increased. Lupton (3) believed that *Septoria* spp. were secondary pathogens that invaded following mildew attack.

The experiments reported here were conducted to determine the relationship between powdery mildew and Septoria leaf blotch on winter wheat.

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GENERAL MATERIALS AND METHODS

Field experiments were conducted near University Park, Pa., in the 1980 and 1981 growing seasons. Fungicides for control of powdery mildew (triadimefon 50 WP, 70 g AI/ha) and Septoria leaf blotch (mancozeb 80 WP, 2.24 kg/ha) were applied to cultivars of varying powdery mildew susceptibility. Plot sizes were 2.4 x 15.2 m and all plots were fertilized with 672 kg/ha of 5-10-10 in the fall and 67.2 kg/ha of NH_4NO_3 in the spring. The experimental design was a split plot with four replications. Disease severity was calculated by evaluating 20 tillers per plot and assessing each leaf per tiller, using the James scale (severity=percent leaf area infected). Plot score was the average of the 20 tillers. Plots were harvested with a combine and yields were calculated. Following analysis of variance, Bayes LSD values were calculated for mean separation.

In 1980, the cvs. 'Potomac', 'Roland', and 'Hart' were used. Respectively, they are susceptible (S), moderately susceptible (MS), and resistant (R) to *E. graminis*. Some of the plots were sprayed with mancozeb fungicide at growth stage (GS) 5 and every 10 days thereafter to control *Septoria* spp. Some plots did not receive the initial spray until GS-7 or GS-9.

In 1981, an experiment was conducted using six cultivars and foliar fungicide applications to control powdery mildew. Triadimefon was applied in the fall, 5 weeks after planting, with some plots receiving an additional application at GS-7 in the spring.

In conclusion, it appears that *Septoria* spp. and *E. graminis* are competitors for leaf area. However, if leaf tissue is previously infected with *Septoria* spp., *E. graminis* will not readily colonize the leaf. These results are in contrast to previous reports suggesting that Septoria is a weak pathogen and is only effective following predisposition of the host.

RESULTS

The results of the 1980 experiment are reported below.

Cultivar	Initial spray	Mildew severity	Septoria severity	Bushels/A
Hart	Control	13.7	21.5	41.7
	GS-5	20.5	6.1	50.7
	GS-7	19.3	15.3	49.5
	GS-9	14.6	20.5	42.0
Roland	Control	10.1	23.8	43.2
	GS-5	18.3	4.1	46.1
	GS-7	18.1	12.6	40.2
	GS-9	11.5	19.5	42.5
Potomac	Control	.3	5.2	40.6
	GS-5	1.2	3.3	39.3
	GS-7	.9	3.4	40.3
	GS-9	.7	3.4	41.2
Bayes LSD (P = 0.05)		1.1	2.0	2.3

The yield of Hart increased when sprays were initiated at either GS-7 or GS-5 while Roland yield increased only when sprays were initiated at GS-5. Both yield increases may have been limited by the increase in powdery mildew since the control of Septoria leaf blotch resulted in an increase in powdery mildew if the fungicide sprays were applied prior to GS-9. The cv. Potomac was resistant to both pathogens and did not respond to the treatments.

In the 1981 experiment results were obtained with all six cultivars. The results are reported below for the cvx. 'Redocat' (MR) and 'Dancer' (S).

Cultivar	Spray treatment	Mildew severity	Septoria severity	Bushels/A
Dancer	Control	8.7	8.6	37.4
	Fall spray	5.4	26.8	56.1
	Fall + spring	2.6	19.9	64.3
Redcoat	Control	1.1	12.9	43.1
	Fall spray	.7	27.1	69.1
	Fall + spring	.4	15.2	71.0
Bayes LSD (P = 0.05)		.4	3.3	3.1

There apparently was a competition between the two pathogens for leaf area. As mildew was controlled, Septoria leaf blotch increased. However, powdery mildew appeared to have the greatest effect on yield.

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FRANK - SPEAKER

Q. Matchett: Do you care to comment on the economics of spraying?

A. The economics has been worked out by an ag economist at the University of Maryland. In most instances, growers would receive an economic benefit if they could afford to control the disease with one spray, but many of our disease problems could not be controlled adequately with one spray. The other problem is that if you do get an increase in septoria like we do in some years, then spraying with a chemical that will only control powdery mildew becomes noneffective. So, in most of our experiments where we do recommend some spraying, we add Mancozeb or Difolatan along with the Triadimefon in order to control both pathogens.

Q. Matchett: Does the Extension Service give out guidelines on when to spray and when to recommend spraying?

A. Yes, and it does vary. In Pennsylvania, we recommend that if you have to make one spray it should be somewhere at approximately growth stage seven, and we instruct the farmers as to what the growth stages are. In Maryland or down into Virginia, however, they feel that the initial spray may have to be put on at growth stage six. The optimum control is two sprays, one at growth stage six and one at nine.

Q. Fehrmann: There is evidence that Bayleton decreases in efficiency over time?

A. I don't know. The only cases that I've ever heard reported are out of England, I believe and those are laboratory tests, but I don't think there are data to indicate that.

Q. Fehrmann: But the length of efficiency was 3 or 4 weeks sometimes. It didn't decrease?

A. Frank: No.

Fehrmann: In The Netherlands, Germany, the United Kingdom, and in several other countries, the efficiency of Bayleton is decreased.

Q. Scharen: I heard you mention that if there was a lot of powdery mildew, you had less septoria; however, I've seen in your area that when I find powdery mildew in an area where I expected to find Septoria nodorum, I'd brush off the powdery mildew mycelium and find pycnidia of Septoria nodorum directly under in the same lesion. Have you ever observed that?

A. I've seen nodorum and mildew coalesce, and it's very difficult to discern which was there first in many of those instances. In many cases, if both organisms show up as we get on growth stage six, and they're both on the leaf, they will both develop. Powdery mildew generally develops a little slower when that happens. However, if you have a significant amount, and generally we're talking about 20-25 percent of the leaf covered with septoria, powdery mildew does not land on that leaf. You'll find it on the leaf above it, but it will not go to the leaf that has 20-25 percent septoria. Sometimes, it may be there, but it is not obvious.

Q. Jones: There was a paper about 10 years ago published by Brian Wheeler and Mark Simpson of Scotland, which describes the exact phenomenon, that which Dr. Scharen mentioned about the mildew growing right on the nodorum lesion.

A. Our powdery mildew in Pennsylvania is generally in small distinct colonies. They are very tiny, and I think if you come in later and look, those colonies are so small you'll see the brown areas in between the mildew and the nodorum.

A BIOASSAY FOR PREDICTING THE RESISTANCE OF WHEAT LEAVES TO SEPTORIA NODORUM

Helen M. Griffiths[1]

Assessing cultivars for resistance to S. nodorum has traditionally been performed by recording the degree of tissue infected (7), the reduction in crop yield (6), and, more recently, using the components of partial resistance (5, 8). All these methods require the examination of large quantities of plant material and do not provide the accuracy which could be obtained if a quantitative method were to be used. Ride and Drysdale (9) described a quantitative method based upon chitin detection. Even though it is a valuable technique for studying many plant--pathogen systems, it has shortcomings in studying the wheat: S. nodorum system (4).

Seitz et al. (10) described a chemical technique based upon ergosterol detection, which has been successfully used in predicting the fungal invasion of cereal grains. The specific absorption of UV light by ergosterol (3) enables it to be characterized and quantified easily, thus, making it ideal for a bioassay.

The aim of the study reported in this present paper was to determine the potential of a bioassay based upon ergosterol detection in assessing the susceptibility of wheat cultivars to S. nodorum.

Plants of the British spring wheat cultivars Highbury (NIAB 2, 'susceptible') and Broom (NIAB 6, 'resistant') were sprayed with inoculum of S. nodorum (1) and covered for 72 hours with polythene bags (modification of Cooke & Jones, 2).

Leaf 3 and leaf 4 were harvested for ergosterol detection on the day of inoculation and then 12, 17, and 27 days after inoculation.

A modification of the ergosterol extraction and quantification method described by Seitz et al. (11) was used in which ergosterol was detected and quantified using high performance liquid chromatography (4).

As no ergosterol was detected in healthy leaf material of either cultivar, the amounts present in the inoculated material can be attributed entirely to fungal invasion of the tissue.

At each harvest time, the amount of ergosterol in the tissue of cv. Highbury was significantly higher ($P = 0.01$) than in cv. Broom (tables 1 and 2).

In addition, the assay was very sensitive with levels of ergosterol as low as 0.09ug per g of tissue being detected (table 1).

The absence of ergosterol in the healthy wheat tissue and the difference in ergosterol levels in the two cultivars after inoculation with S. nodorum indicates that this assay has considerable potential in assessing wheat cultivars for resistance to this pathogen.

This assay could be of value to the plant breeder as it fulfills many of the desired criteria, namely a rapid, reproducible, sensitive method using most of these requirements, although, the initial stage (saponification) is somewhat tedious and could limit the general use of the assay. However, the method could certainly be of great value for screening for resistance to S. nodorum at the final stages of a breeding program when fewer cultivars are being considered and precise results essential.

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Table 1.--The ergosterol content of tissue from cvs. Highbury and Broom at several times after inoculation.

	Ergosterol content $\mu g/g^{-1}$ tissue x 10^{-3}		
	Harvest 1 (12 days)	Harvest 2 (17 days)	Harvest 3 (27 days)
Highbury (NIAB 2)	90 ± 52	230 ± 16	450 ± 30
Broom (NIAB 6)	0	170 ± 20	290 ± 46

Table 2.--Summary of analysis of variance.

	D.F.	Mean square	Variance ratio
Total	17		
Replicate	2	0.00035	0.13 N.S.
Combination	5	0.07328	29.20 ***
Cultivar	1	0.0512	20.40 **
Harvest	2	0.1538	61.30 ***
Harvest x cultivar interaction	2	0.0038	1.50 N.S.
Error	10	0.00251	

*** P = 0.001
** P = 0.01
* P = 0.05
N.S. = Not significant.

GRIFFITHS - SPEAKER

Q. Tomerlin: Did you observe any sporulation with your pycnidia in your harvest 3?

A. No.

Q. Marshall: Was there any typical reaction at all in the 27 days? Were there differences?

A. Yes, I did actually score the plants for resistance at least two times, and at that time there was a difference between the two varieties.

Q. What were the results at stage four?

A. We haven't had it scored, unfortunately. It would be nice to carry on.

Q. I observe they go up after three.

A. Yes, they do.

Q. Scharen: You mentioned you wouldn't want too many replicates of too many of these experiments. What sort of time frame does it take to do one of these analyses?

A. It depends if you've got your own lab facilities. If you do, I guess you could go through it in a couple of days.

CHEMICAL CONTROL OF SEPTORIA NODORUM IN WHEAT

H. Fehrmann[1]

Worldwide, the economic importance of the Septoria spp. in wheat increased considerably during the sixties and the early seventies. In one respect, this was due to the introduction of newly bred dwarf and semidwarf varieties, mainly in warm climate countries. On the other hand, the introduction of the haulm-stabilizing chemical chlorocholine-chloride (CCC) in Europe was succeeded by a tremendous increase in Septoria attack. In most cases, this is due to S. nodorum, but S. tritici may be involved or even dominate.

This situation may be explained in two ways: Shortening the haulm facilitates the movement and rain-splash of the conidia from the lower to the upper leaves and to the ears, thereby increasing flag leaf and ear attack. Moreover, haulm stabilization enables conventional nitrogen dosages to be doubled or even tripled up to 200 kg/ha. This in turn favors powdery mildew, which again may act as a step-maker for succeeding Septoria attack.

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CHEMICAL CONTROL BY SPRAYING

Mainly from the financial profits from powdery mildew control in barley in the late sixties, farmers became aware of the advantages of spraying fungicides against cereal pathogens. Chemical control of glume blotch commenced about 1972, and control of eyespot, in 1974. Financial profit from chemical protection in Europe is based on a reasonable relationship between financial input and net return. This is guaranteed by high fixed prices for grain on the one hand, and by higher yield per hectare on the other--up to 10,000 kg/ha or higher.

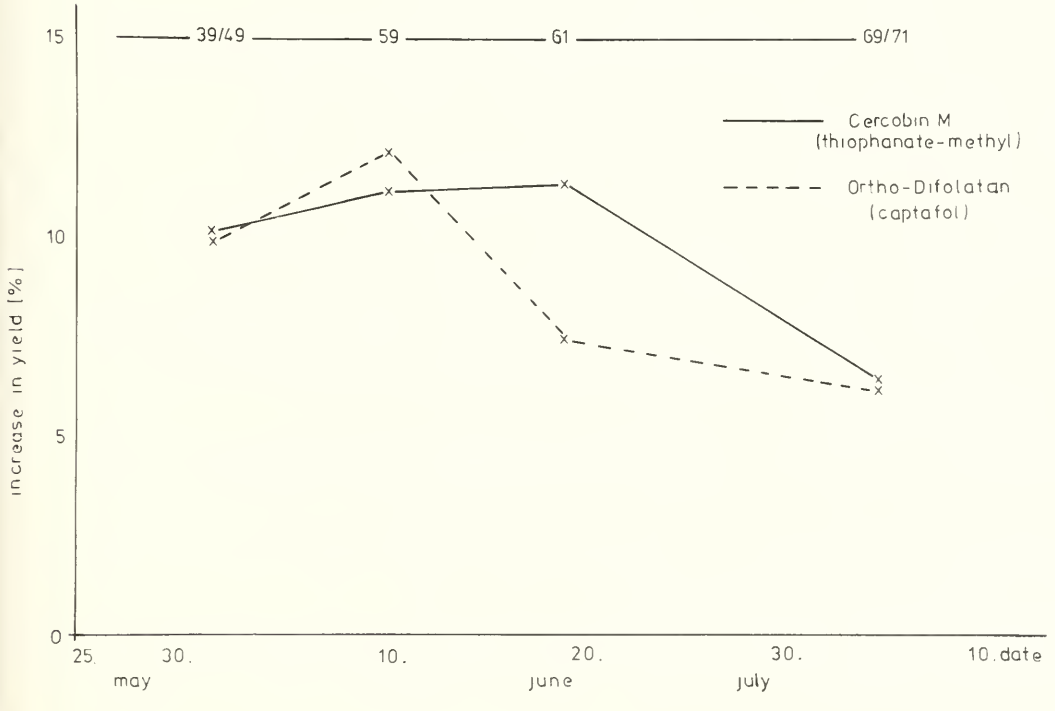
Conventional Fungicides

Chemical control of ear diseases was first achieved with MBC-type fungicides (7, 22). Increases in yield were profitable in cases of moderate or more severe ear attack by glume blotch and/or powdery mildew. Nevertheless, at least in West Germany, toxicological scruples led to the withdrawal of the official usage of MBC-type fungicides on ears. Since then, captafol is mostly used for the control of glume blotch (24, 25, 26). Application is normally done at heading, when about 75% or more of the ears have appeared (fig. 1).

Recent Development

The main disadvantage of captafol and other protectants is the critical timing required, being limited

Figure 1.--Increase in yield of winter wheat after spraying a curative (thiophanate-methyl) and a protective (captafol) fungicide, respectively, at different plant states; before heading; end of heading; beginning of flowering; after flowering. Average data from four field experiments, Goettingen.



to a short period after heading. Moreover, this chemical is poor in controlling leaf attack by the Septoria spp.

To a certain extent, this problem was overcome by the introduction of new ergosterol-biosynthesis inhibitors such as prochloraz (Sportak) and propiconazole (Tilt) (5, 8, 12, 13, 27, 28).

Table 1 shows some typical results with prochloraz in a variety moderately resistant to eyespot. Prochloraz and--for comparison--carbendazim (Derosal 60%), triadimefon (Bayleton), and captafol + triadimefon (Bayleton DF, 65 + 6.25%) were applied at three successive stages of the plant, i.e. at two-node-stage up to flag leaf just visible (GS 32/37), followed by GS 37 or 39 (preboot stage) and at heading (GS 51/59), respectively.

The increases in yield after the first and second treatments to a limited extent could be due to some eyespot control by prochloraz; attack by powdery mildew and glume blotch was only weak. This allows for the interpretation that increases in yield are mainly due to chemical control of S. nodorum leaf blotch. Assessments were made according to James (17).

In two other experiments, propiconazole (Tilt, Desmel) was applied at different dates on two different varieties (table 2).

Combined spraying at two different dates was more suppressive on S. nodorum leaf blotch and more effective on yield than a single treatment. Glume blotch infection was light, and assessment data after chemical control were not significantly different from the untreated control. Again, the results demonstrate a reasonable, but not an eliminating effect on leaf attack. The effectivity depends very much on right timing. Until now, practical experience has shown, that treatment at preboot or boot stage is most likely to be economical if the attack on the lower leaves is heavy, moderate on the third leaf, and already visible on the second leaf. As yet, there are no definite results and rules for this, and the situation changes with the varieties.

In two other experiments with different varieties, artificial inoculation with S. nodorum led to decreases in yield by about 20% (table 3). The inoculum was sprayed after heading and led to severe glume and leaf blotch. A single protective application of prochloraz five days before inoculation was less effective than curative treatments one week after inoculation. A combination of two sprayings at both these dates was the most effective, but was not economical as compared with the single postinfectional treatment at the beginning of milk development (water-ripe stage, GS 71). A single late treatment (stage 75, medium-milk) was useless.

To summarize, the introduction of new curative fungicides for the control of S. nodorum has several

Table 1.--Influence of prochloraz and other chemicals on yield and Septoria nodorum leaf blotch in winter wheat cv. Caribo (Goettingen, 1981). Leaf blotch assessed as percent of leaf area affected.

Fungicide	Application Date stage		Yield		S. nodorum leaf blotch Third leaf (GS 69/71)	Second leaf (GS 75)
			Kg/ha	Percent		
Untreated			3.810	100.0	25.1	18.7
Prochloraz	5/18	32/37	4.310	113.2	18.4	13.7
Carbendazim	5/18	32/37	4.080	107.2	21.1	18.9
Prochloraz	5/25	37/39	4.400	115.4	11.9	15.4
Triadimefon	5/25	37/39	4.000	105.0	26.5	11.1
Prochloraz	6/11	51/59	3.920	103.1	18.5	6.3
Triadimefon + captafol	6/11	51/59	4.110	108.0	24.3	11.9

Application rates: carbendazim (Derosal 60%) 0.18 kg/ha a.i.
triadimefon (Bayleton 25%) 0.125 kg/ha a.i.
prochloraz (Sportak 40%) 0.480 kg/ha a.i.
captafol + triadimefon (Bayleton DF, 65% + 6.25%) 1.3 + 0.13 kg/ha a.i.

Table 2.--Influence of propiconazole (Desmel at 0.5 l/ha) on yield and S. nodorum leaf blotch. Average data from two winter wheat field experiments, 1981 (cv. Caribo and Diplomat). S. nodorum leaf blotch was assessed at GS 69/71 (June 24) and GS 75 (July 6).

Application Date	stage	Yield		Percent leaf blotch area: Third leaf (69/71)	Second leaf (75)
		Kg/ha	Percent		
(Untreated)		4.270	100.0	26.7	25.1
5/25	37/39	4.260	99.8	14.3	9.4
6/11	51/59	4.500	105.4	21.8	9.4
5/25+ 6/11	37/39 51/59	4.910	115.0	11.8	5.2
5/12+ 6/11	31/32 51/59	4.680	109.6	9.8	7.6

Table 3.--Influence of prochloraz on yield, S. nodorum leaf blotch in two winter wheat experiments with cv. Kormoran and Caribo, 1982, with artificial inoculation.

Application Date	stage	Yield		Leaf blotch (stage 75, July 1) Second leaf	Third leaf	Glume blotch (stage 75, July 1) July 6	July 15
		Kg/ha	Percent	Percent			
Untreated, Uninoculated		7.580	100.0	11.1	28.3	1.2	3.0
Untreated, Inoculated		6.130	80.9	44.8	61.0	43.1	78.6
6/7	51	6.650	87.8	23.0	35.8	36.6	70.6
6/18	71	7.230	95.4	9.8	26.1	23.5	55.3
6/7+ 6/18	51 +71	7.370	97.2	8.6	17.0	16.1	50.6
7/2	75	6.430	84.8	44.4	60.4	37.0	71.4

advantages and disadvantages, as compared with the traditional spraying of captafol or other protectants:

Advantages:

* In addition to glume blotch, economic control of S. nodorum leaf blotch was also achieved,
* Timing for the control of glume blotch is more flexible,
* The newly introduced chemicals are broad-spectrum fungicides covering powdery mildew, rusts, and other late diseases.

Disadvantages:

* The new curative chemicals are more expensive than protectants,
* There is speculation of fungicidal resistance.

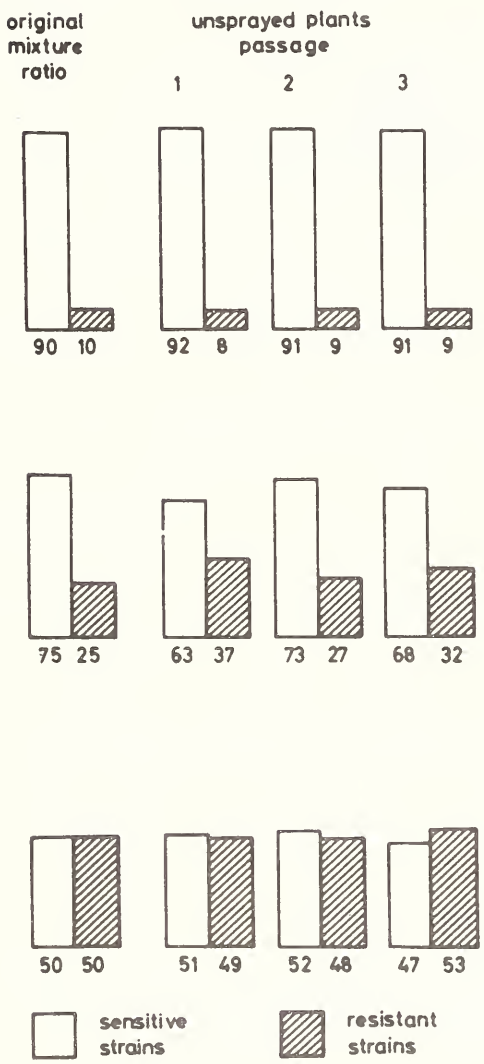
Danger Of Fungicidal Resistance

Under field conditions with up to four applications per year, S. nodorum strains resistant to MBC or edifenphos accumulate in field populations on a negligible scale (15). MBC resistant and sensitive

strains are equally fit, while edifenphos resistant strains are less competitive than sensitive ones in mixed populations under conditions without fungicide selection pressure (fig. 2; 16). Until now, there have been no reports of MBC failures in glume blotch control. On a long-term basis, however, one has to be aware of fungicide resistance in the field even under circumstances of extremely low selection pressure (9).

Limpert, (pers. comm.), in Germany, has shown that triadimefon or triadimenol sensitivity of field populations of barley powdery mildew may decrease to some extent over a number of years. This may decrease the length of efficacy within the plant. Triadimefon and triadimenol are ergosterol-biosynthesis inhibitors, such as prochloraz and propiconazole, and cross-resistance has been reported for several fungi within this group of fungicides (4 and Buchenauer, pers. comm.).

Figure 2. Survival of carbendazim resistant strains of S. nodorum in mixed populations during three successive passages on wheat plants. Plants remained unsprayed.



For these reasons, introduction of new one-site inhibitors into practice should involve as much caution as possible to avoid resistance problems.

CHEMICAL CONTROL BY SEED TREATMENT

There is a close correlation between the percentage of S. nodorum infected seed and the number of infected seedlings (14).

Chemical control by seed treatment is possible with mercury, MBC-type fungicides, and others. By this way, seedling emergence can be improved considerably, but at harvest no differences may be observed between the plots with or without seed treatment (10, 11). Bateman (3) did not find any differences in S. nodorum leaf blotch after foregoing seed treatment with mercury, carboxin, maneb, guazatin, or thiabendazole. In contradiction to these findings, Jenkyn and King (20) reported that mercury treatment of wheat seed resulted in improved seedling emergence, higher yield, and less S. nodorum leaf blotch on the flag leaves. Obviously, previous reports on this important topic are not conclusive enough. Treatment of S. nodorum contaminated seed may result in less leaf or even glume blotch and a higher number of ears per square meter under certain conditions.

INTEGRATED CONCEPTS

The most promising way for integrated control is breeding for resistance. This topic is covered by other papers of this meeting. However, decision making in chemical control should involve knowledge about the susceptibility of a variety, the promoting effect of CCC, and meteorological conditions, such as rainfall, temperature, relative humidity, and length of leaf wetness. A farmer should be aware of the influence of agricultural procedures on the inoculum potential, such as crop rotation, depth of ploughing or minimum tillage, burning of stubbles, and others (6). Of course, the best means of helping a farmer in decision making would be a profound knowledge about the relationship between disease incidence at spraying and the crop loss to be expected from it.

Crop-Loss Appraisal

Reliable data for economic threshold values for chemical control are difficult to achieve. They depend on many factors, such as variety, weather conditions, and the inoculum potential available. Therefore, it is nearly impossible to develop something like standard threshold values as an aid for decision making at spraying time. If this coincides with the time of heading, the picture still is somewhat clear. If applications are made at different times, e.g., at boot stage and at flowering, the situation becomes confused.

When glume blotch assessment was made at flowering (GS 61/69) in two spring wheat varieties ('Kolibri and 'Selpek'), the correlation coefficient for the relationship between single-grain weight at harvest and glume attack was quite high--between 0.81 and

0.91. Corresponding data for single-ear yield varied between 0.70 and 0.86, if glume blotch data or S. nodorum leaf blotch data from the flag leaf and the second leaf were used for calculation (1). According to these results, between 50 and 83% of the variance for yield loss could be explained by S. nodorum attack on either plant organ--the head or flag leaf. In association with powdery mildew on the ears or with take-all, the data are modified (figs. 3, 4, and 5). For cv. Selpek, about 1% glume blotch (necrotic glume area) caused 1% loss in single-ear yield; 1% S. nodorum leaf blotch on the flag leaf caused about 0.4% loss in single-grain weight, as was shown for both cultivars. On the other hand, only about 0.5% loss in single-grain weight could be attributed to 1% glume blotch in cv. Diplomat. In the latter case, data were modified by eyespot.

To serve as an aid in plant protection, the value of such data is relatively poor. Here, assessments are made at flowering, even after the optimum stage for application of protectants such as captafol. Employing earlier assessment data for crop-loss appraisals as a tool for decision making, correlation coefficients are too small. The relative contribution of the attack of different plant organs in explaining yield losses changes from one field to the other (fig. 6). Again, this implies that fixed incidence values for economic threshold values are difficult to elaborate.

Septoria Prognosis Systems

In the past, there have been several efforts to establish forecasting or warning systems for the chemical control of S. nodorum; mostly, they were derived from meteorological measurements. At present, a method worked out by a group in Weihenstephan, Germany, (21) is quite popular, but still under development. In this country, about 35% of the wheat acreage is free from attack by S. nodorum, while about 20% suffer from severe attack. For the warning system, for a period of 20 days before and after heading, daily readings are taken for temperature and relative humidity at 2 p.m. and totaled for certain time intervals (mostly pentades). If certain threshold values are reached or exceeded, and if some modifying requirements are met, such as location, variety, and crop rotation, the farmer is advised to spray. The system works on a pilot basis in southern Germany, and computation is performed by a central computer of the Bavarian Ministry of Agriculture. Farmers or advisory institutions using the system have to be connected on-line. There are efforts to employ a specifically devised instrument for this procedure, which enables meteorological recordings and computations in the field (29).

Variety Mixtures

Recently, there is a very popular trend in Europe of escaping disease problems by growing mixtures of cereal varieties. This is practiced in England and Denmark mainly to control barley powdery mildew without using chemicals. As a pathogen, midlew is specialized to certain varieties by pathotypes, and mixing varieties with a different pattern of resistance genes slows down the progress of a field epidemic.

S. nodorum is an unspecialized pathogen with no variety or major resistance gene specific pathotypes known. Nevertheless, Jeger et al. (18, 19) claim from theoretical considerations and from computations, that growing mixtures of septoria-resistant and susceptible varieties should reduce disease development. In field experiments with two spring wheat varieties ('Kolibri' and 'Maris Butler'), it was shown that disease progress in the mixture was almost the same as in a pure stand of the resistant

Figure 3.--Effect on single-ear yield by glume blotch and powdery mildew on ears, cv. Selpek (1).

Figure 4.--Effect on single-grain weight by glume blotch and eyespot, cv. Diplomat (1).

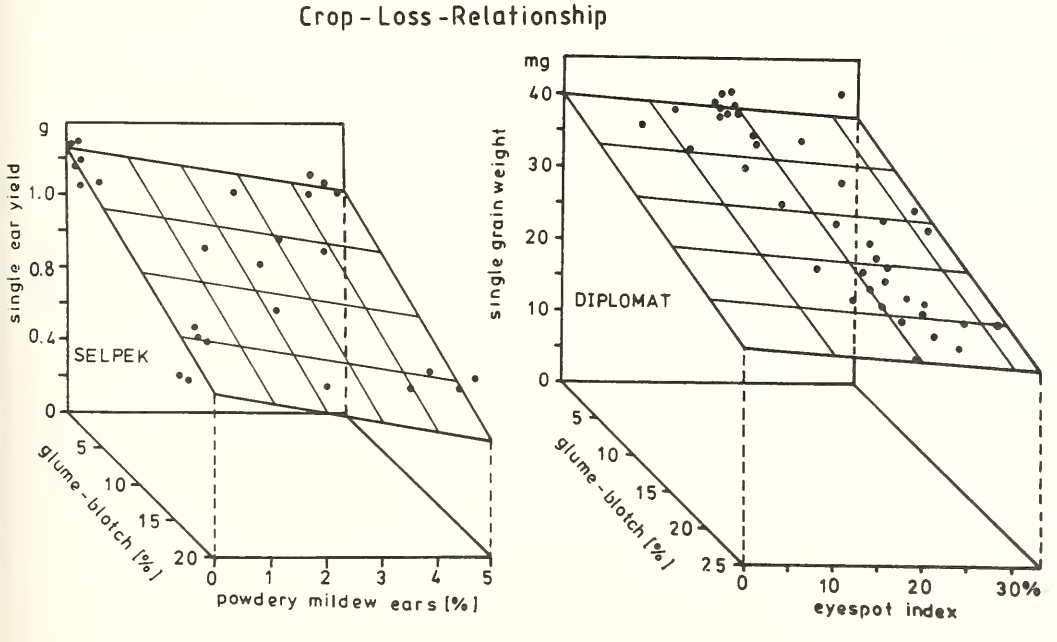


Figure 5.--Effect on single-grain weight of S. nodorum leaf blotch, take-all and powdery mildew, cv. Selpek Kolibri (1).

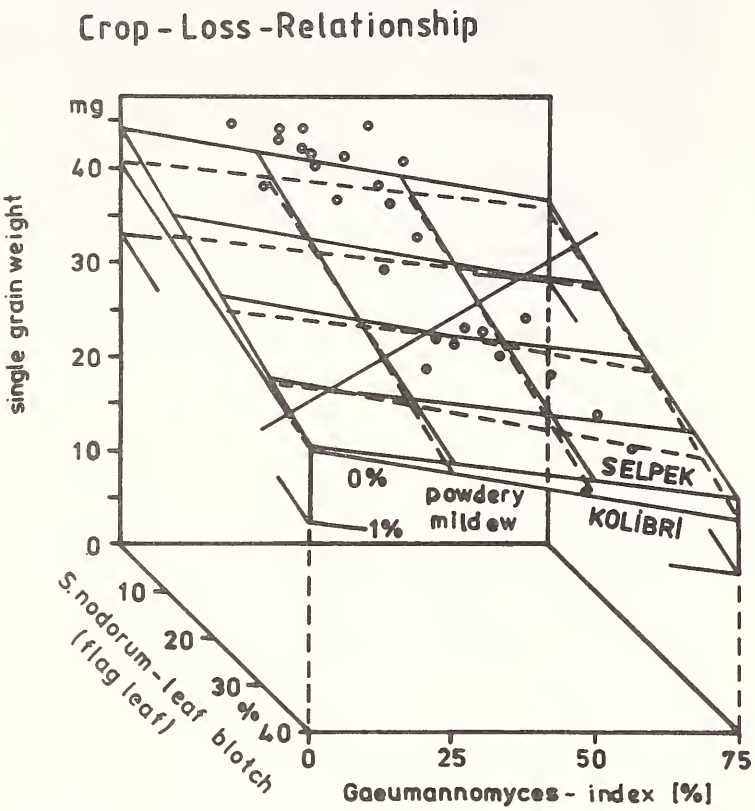
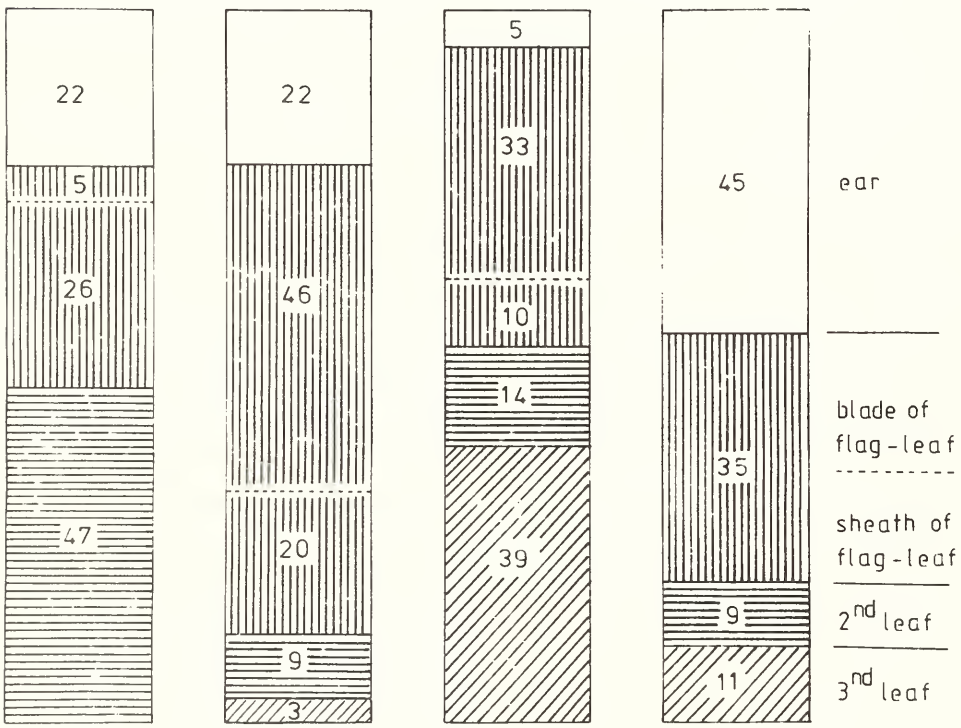


Figure 6.--Contribution of S. nodorum attach on different plant organs to loss in single-grain weight in four different wheat varieties/locations (2).



variety. The disease level, however, was fairly low. The published data on yield are convincing.

The system is based on a multigenic reaction of the host plant, and not a single-gene reaction as is the case with powdery mildew of barley. Here, factors such as infection frequency (number of lesions developing from a given spore suspension), sporulation rate, and length of the latent period seem to play an important role.

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FEHRMANN - SPEAKER

Q. D. Gareth Jones: The data you have for the two chemicals on seed control didn't seem to be very successful. Do you care to qualify that?

A. They were not successful. I only wanted to show you the dependency that in principle is possible, that in the type of the grains could be expected but you cannot control seed infection.

Q. Ariano Prestes: Do you have results combining prochloraz and propiconazole?

A. No, we have not made this in the same experiment. They were different experiments, but I do not think there is much difference. There may be experiments going on with these two chemicals being tested at the same time.

Q. Prestes: What should be done to avoid resistance in septoria to fungicides?

A. This is a difficult question. In our country, where the application of systematic fungicides is quite frequent, there are three or sometimes four applications of the same type of chemical. The selection pressures in the varieties could increase so much that you could expect that this could bring problems within a few years. After a few years of three or four applications of MBC in the field, in two cases at least, problems came up with fungicidal resistance. In one case, also, in the experiment after 7 years of two or three applications a year, we would have resistance in the population. So, to escape this problem of fungicidal resistance the farmer should always exchange his wheat varieties after 1 to 3 years. The conventional fungicide should be exchanged with the systematic or ultimately this type of fungicide with other types of fungicides. This is a way to escape these problems, but, of course, it is very difficult to convince the farmer because of his background.

Q. L. R. Nelson: Have you seen any stimulating effects of fungicides on different varieties?

A. I couldn't say much about differences in varieties, but some fungicides tend to extend periods of green leaves. These are only observations. I think we have data that these chemicals also have an effect on such fungi as saprophytic microflora of the leaves.

Q. John E. Watkins: We have used them both on wheat and on blue grass. On wheat, I've never seen anything killed on the bottom parts, but on blue grass, fungicide application did prolong the time tissue stayed green on the bottom leaves of the plants.

Q. Robert Hosford: Do you know if the chemical is affecting the fungus or the host?

A. The critical mode of action of both these chemicals is that the synthesis of ergosterol is inhibited through these channels. So, another effect on the plant would be completely secondary.

Q. Hailu Gebre-Mariam: Do you have any experience in Septoria nodorum or yellow rust?

A. No, yellow rust in our country is not a regular problem. But as far as I know from the United Kingdom, it is possible to control Septoria nodorum and yellow rust with both these chemicals. The influence on rust is minimal; so, for the control of rust you have to increase the amount of chemicals applied on the fields, but in principle you can control both with these chemicals.

Q. Prem Kharbanda: In your experiments, you got better control when you applied fungicides as correctives rather than protectives. In your experiments, you got better control on one field where the infection had already started. Is there any reason why you have low yield variance to control the fungus after the disease has started? Is there a difference between fungicides or the time of the application?

A. The curative effect on Septoria nodorum seems to be better according to the results we have, but the curative effect seems to be only a little bit better than the protective effect. This could be due to some breakdown of the chemical between application and action, but I would not say that our results with the protectant are worse than with the correctives.

Q. Prem: You could apply the same fungicides twice as a corrective and again as a protective?

A. Yes, of course, then you are successful, but this is very expensive. Prices are very high. You have to harvest, I would say 150 to 200 kg/ha more only to cover the price of the chemical.

FUNGICIDAL CONTROL OF SEPTORIA NODORUM ON WHEAT WITH PROPICONAZOL (TILT)

L. R. Nelson and George Philley[1]

The fungicide propiconazol (Tilt, CGA-64250) was applied to four wheat varieties over 3 years at Overton, Texas. Application of Tilt significantly (0.01 level) increased yields over the control in 2 of 3 years and reduced glume blotch (caused by Septoria nodorum Berk.) rating in all 3 years. Yield response with individual varieties varied with years. However, Tilt increased yields on McNair 1813 more than other varieties. Yields for McNair 1813 were increased 45 and 48% for the treated plots in 1980 and 1982, respectively. The mean increase in yield due to the fungicide treatment over 3 years and all varieties was 14%. These results indicate that in 2 of 3 years a fungicidal treatment was successful and may have been cost effective.

OBJECTIVE

Our objectives were to determine the effectiveness of Tilt in controlling glume blotch and increasing wheat yields and to determine the interaction between genotype and response to Tilt.

PROCEDURE

The study was conducted from 1979 through 1982 at the Texas A&M University Agricultural Research and Extension Center at Overton, Texas. A complete tillage system was utilized with planting in late October. Preplant fertilization was 60 lb each of N, P_2O_5, and K_2O/ac (67 kg/ha) applied broadcast and disked into the soil. Nitrogen was topdressed as ammonium nitrate in February each year at a rate of 60 lb N/ac (67 kg/ha). The seeding rate was 75 lb/ac (84 kg/ha). Each plot was six rows wide, spaced 8 inches apart (20 cm), and was 10 ft long (3 m). The test was applied in a randomized complete block statistical design with four replications. Plots were trimmed to 8 ft in length (2.44 m) prior to harvest. Three of the center rows were harvested for grain. The varieties in the study were Coker 68-15, McNair 3069, Tx-73005 (tolerant to S. nodorum), and McNair 1813 (susceptible). The fungicide application was made twice each year about 1 month apart in early April in the boot stage (10.0 growth stage) and in early May in the milk stage (10.6 growth stage). Tilt was applied with a solo mist blower at a rate of one-half pint or 100 g a.i./ac (247 kg/ha). Wheat was inoculated in the field with S. nodorum only during the third year of the study; natural infections were utilized during 1980 and 1981. Glume blotch ratings were taken on a scale from 0 to 9 where 0 was nondiseased plants.

RESULTS AND DISCUSSION

Environmental conditions in 1980 and 1982 resulted in severe epidemics of S. nodorum and significant yield reductions. Disease levels in 1981 were light with little yield reduction due to S. nodorum (table 1). The Tilt treatment significantly (0.01 level) increased yields by 20 and 21% over all varieties in 1980 and 1982, respectively. Glume blotch ratings were significantly reduced (0.01 level) in all 3 years with fungicidal application.

Varieties were not significantly different for yield. However, variety x fungicide interaction for yield was significant at the 0.07 level over years (table 2). McNair 1813, a susceptible variety, had a 28% yield increase when Tilt was applied. The glume blotch ratings indicate we obtained good control of the fungus. Somewhat surprising, the mean glume blotch rating for the untreated McNair 1813 wheat was 4.1, which was the lowest rating among the four varieties, yet this variety responded in increased yield more than all other varieties.

The data from 1980 (table 3) and 1982 (table 4), indicate good yield responses occurred. In 1980, Tilt increased yields of McNair 1813 and Coker 68-15 by 45 and 21%, respectively. The yield of untreated McNair 1813 was only 3024 kg/ha (45 bu/ac) but increased to 4375 kg/ha (65 bu/ac) when treated, which would have more than paid for the cost of spraying the wheat. The glume blotch ratings indicate an effective control of the

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Table 1.--Yield and glume blotch rating of control versus Tilt sprayed treatments averaged over varieties for 3 years.

	Yield kg/ha			Glume blotch rating	
Year	Control	Sprayed	Percentage of increase	Control	Sprayed
1980	3132	3750**	20	6.2	2.4**
1981	3149	3264	4	4.1	2.9**
1982	1921	2325**	21	4.6	2.6**

**Significantly different from the control at the 0.01 level.

Table 2.--Yield and glume blotch ratings of control versus Tilt sprayed wheat for 4 varieties averaged over 3 years.

Variety	Yield kg/ha		Percentage of increase	Glume blotch rating	
	Control	Sprayed		Control	Sprayed
Coker 68-15	2957	3158	7	5.3	2.3
Tx-73005	2654	2950	11	5.8	3.3
McNair 3069	2722	3011	11	4.6	2.8
McNair 1803	2601	3340	28	4.1	2.1
Mean	2735	3111**	14	4.9	2.6**

**Indicates means (control vs. sprayed) averaged over varieties for yield and glume blotch were significantly different at the 0.01 level.

Table 3.--Yield and glume blotch rating of 4 varieties on control versus Tilt sprayed treatments for 1980.

Variety	Yield kg/ha		Percentage of increase	Glume blotch rating	
	Control	Sprayed		Control	Sprayed
Coker 68-15	3271	3951	21	6.3†	1.0
Tx-73005	3017	3397	13	6.0	3.5
McNair 3069	3205	3284	2	7.0	3.75
McNair 1813	3024	4375	45	5.5	1.3
Mean	3132	3750**	20	6.2	2.4**

**Indicates means (control vs. sprayed) were significantly different at the 0.01 level.

†There was a highly significant interaction between varieties x fungicide treatment for Septoria rating.

disease; however, a significant (0.01 level) interaction for variety x fungicide treatment indicated that the treatment controlled S. nodorum better on some varieties than on others (Coker 68-15 and McNair 1813).

In 1982 (table 4), disease severity was high, not only for Septoria, but also for leaf rust and powdery mildew which confounded the results. Nevertheless, Tilt controlled all three diseases, but the yields did not respond as well as expected. McNair 1813 and 3069 had yield responses of 48 and 26%, respectively, due to the fungicide treatment.

These results indicate that in 2 out of 3 years, the use of Tilt to control fungus pathogens, in particular Septoria nodorum, likely would have been worthwhile. In 1981, disease severity levels were very low. With the variety McNair 1813, a fungicide treatment would have been worthwhile and probably cost effective.

Table 4.--Yield, glume blotch, and leaf rust ratings on 4 varieties on control versus Tilt sprayed treatments for 1982.

Cultivar	Yield kg/ha		Percentage of increase	Glume blotch rating		Percentage of leaf rust	
	Control	Sprayed		Control	Sprayed	Control	Sprayed
Coker 68-15	2098	2269	8	5.5†	3.3	80†	3
Tx-73005	2097	2352	12	6.0	2.3	60	4
McNair 3069	1873	2359	26	3.5	2.3	35	3
McNair 1813	1613	2387	48	3.3	2.5	35	3
Mean	1922	2325**	21	4.6	2.6**	53	3**

**Indicates means (control vs. sprayed) were significantly different at the 0.01 level.

†There was a significant variety x spray interaction for both Septoria rating and for leaf rust.

NELSON - SPEAKER

Q. Maarten van Ginkel: The scale that you were using, was it the Saari-Prescott scale?

A. The way I do that, I'll take the ratings two or three different times during the season. I like to take the ratings of the plot by walking by, taking an overall view in my mind of the plot, and assigning a rating. I find that that works out very well for me.

Q. Maarten van Ginkel: Did you rate on the height of disease or type of reaction?

A. In our area, even in years when it is not severe, the disease always gets on the head. So maybe height is not of that much importance. I do pay quite a bit of attention to the amount of lesions on the glumes. Probably, that is most important in our rating.

Q. Gregory Shaner: In the year that mildew was a problem, do you think it became severe enough to confound your septoria data?

A. I don't think so. We didn't take a test count though, so it could be partially due to that. I don't know that there was a big difference between varieties. Some of those varieties are more resistant to mildew than others, so I can't really answer that. It looks like mildew probably was the most suspect thing for keeping those yields down so much, unless you get some root rot diseases there.

Q. F. R. Sanderson: When did your infection first come in? As you put on the spray aimed to control mildew, what effects did it have on the septoria?

A. We are trying to answer some of those questions. We do have some studies on the way presently where we are applying the fungicides at different times, some of them quite early, and hopefully we will have more information on the way. I suspect that we get septoria very early on the lower leaves. It is going to be interesting to know what time we need to start controlling the disease.

Q. Why should you put on many applications if you've only got one source of inoculum? You've got no further amounts coming in from other crops.

Comment from Fehrmann: You cannot suppress it. You have so much inoculum potential left that you have to do it for a second time.

Q. Sanderson: Even then you are putting the chemical on too late. In the New Zealand situation, here I am talking about Septoria tritici, it is not all that effective on speckled leaf blotch if it is put on after the epidemic has started to build up. In one case in New Zealand, the chemical was put on before the speckled leaf blotch symptoms even started to show up, and in that situation the farmer got 100 percent control of speckled leaf blotch. I'm suggesting if you apply early enough in your epidemic you will get sufficient control.

A. I don't think we would get sufficient control. I think in our area that we don't get a great deal of build up until the milk stage. At that stage, if you do not have chemical protection the disease explodes. If you have several rainy days, it gets all over the plants. I would think if you hadn't sprayed within 3 weeks, there would be enough inoculum present that it would attack the plants.

Q. Shaner: When you say it explodes after rainy weather, how long does it take for symptoms to appear?

A. About a week. We have had situations where the foliage does not dry out for several days. About a week after that, it can kill the susceptible plants completely and they don't produce anything whatsoever. Basically, they turn brown and die.

Q. Gareth Jones: The data in the Ukraine states that with the two fungicides you used, symptoms appear after 8 to 9 days. You can never eliminate the disease completely. Do you have any details of how these varieties interact?

A. I don't have that. I will be getting the data on which yield components may be affected more than others. I don't have any data on the latent period at this point.

Q. Jim Frank: How do you apply your fungicides? Ground rig or air?

A. This was applied with a Solo mist blower.

Comment by Frank: Later applications in many cases might result in spreading glume blotch. The grower does not want to go into the field that late. A lot more damage is done by driving through the field at growth stage 10 than by driving through the field at growth stage 8. In many cases, you are better off going in earlier with a heavier rate rather than going in later and having to drive through the fields because they suffer a lot more damage driving through the fields later on.

Nelson: This could be done by aerial application.

Comment by Fehrmann: I think that the chemical protection in your country is much better than in our country. The situation 10 or 15 years ago in Europe was that the farmers went into the fields and sprayed the fields until just before harvest, but then they switched over to tram lines in practically every field. In the neighboring rows, there is a border effect. Tram lines have become popular in the United Kingdom and Germany. I think the economic situation in your country is different as far as chemicals are concerned. You would apply much more fungicides if it would be more price effective.

Q. Prestes: It is very important to start spraying early, but in Brazil, we spray for two reasons. One is diseases are very intense because of heavy moisture. The second reason is that, in general, varieties are susceptible. We have to spray even though spraying in the elongation stage means that we have to spray again at least once after heading. I think that the level of resistance in varieties is very important to the level of control.

EFFECTIVE CHEMICAL CONTROL OF FOLIAR FUNGAL DISEASES OF WINTER WHEAT IN NEBRASKA

John E. Watkins, Ben L. Doupnik, Jr., and Michael G. Boosalis[1]

Two leaf spotting diseases and one rust disease commonly cause damage to winter wheat grown in Nebraska. Septoria leaf blotch (*Septoria tritici* Rob. in Desm.), tan spot (*Pyrenophora trichostoma* (Fr.) Fckl.), and leaf rust (*Puccinia recondita* Rob. ex Desm. f. sp. *tritici*) are the major foliar diseases of winter wheat in Nebraska (4).

Annual losses in the State's winter wheat due to these diseases are usually less than 5 percent because the prevailing hot, dry, windy weather during the post-boot stage is not favorable for their development on the flag and flag-1 leaves.

Occasionally, as in 1977, 1981 and 1982, the weather from April through June is ideal for leaf spot and leaf rust development (2, 3, 4, 5). In 1977 and again in 1981, the incidence of leaf rust and leaf spots was moderately high with *P. trichostoma* the predominant leaf spotting fungus. The years from 1978 through 1980 were relatively dry and not conducive to severe leaf spot or leaf rust outbreaks.

In 1982, however, unusually severe disease losses were incurred by wheat growers in central and eastern Nebraska. The diseases involved were wheat scab (*Fusarium* spp.), Cephalosporium stripe (*Cephalosporium gramineum* Nisik & Ikata), Septoria leaf blotch, tan spot, and leaf rust. Late spring and early summer weather was unusually cool and wet over much of the State and was the primary factor contributing to the impact of these diseases on production losses. For example, at Clay Center in south central Nebraska, 37 days of measureable precipitation were recorded between May 1st and June 30th. Wet, cloudy, calm weather kept much of the eastern and south central Nebraska wheat crop continuously wet during the flowering and grain filling period. This brought about an influx of foliar diseases. Septoria leaf blotch and leaf rust were predominant in eastern and south central Nebraska with tan spot more prevalent in the west.

Wheat became infected with Septoria in the fall of 1981. These infected leaves apparently provided spring inoculum because Septoria began to appear in early April of 1982. Septoria continued to move upward on the plant and combined with severe leaf rust, prematurely killed the flag and flag-1 leaves.

Extension plant pathologists recommended that fields be aerially sprayed with mancozeb (Dithane M-45 or Manzate 200) at boot stage. Those fields that were treated showed substantially less injury to the flag leaf than surrounding nonsprayed fields. Table 1 shows the results of the 1982 foliar fungicide trial at the University of Nebraska's South Central Station near Clay Center, Nebr., and illustrates the effectiveness of fungicides in disease control, higher yields, and increased 1000-kernel weights (5). Plots treated with Tilt (CGA-64250; Ciba-Geigy), Bayleton 50W (Mobay), the high rate of RH-5781 (Rohm & Haas) with the spreader between 20 or both rates of RH-5781 in combination with Dithane M-45 showed less Septoria leaf blotch, except for Bayleton, and lower leaf rust severities. Yields from these plots were from 36 to 41 percent higher than the yield of the nontreated check. Plots receiving one or two applications of Dithane M-45 showed less leaf rust but leaf spot ratings were equal to or greater than those of the check plot. Yields of these plots were higher than those of the check plot, but the differences were not significant.

The 1982 wheat growing season was unusual in that yield differences between certain treated and untreated plots were large enough to show an economic advantage to spraying. At a selling price of $4 per bushel and treatment costs estimated at $18-20 per acre, the 15-bushel-per-acre increase obtained by the RH-5781F + Dithane M-45 treatment would have brought an approximate net return of $40 per acre. In most years, the yield differences between treated and untreated plots have been small and spraying would not have returned a net economic gain. Because of this yearly variability, application of foliar fungicides to winter wheat for leaf spot and leaf rust control is not recommended as a routine practice. The decision on whether to spray is based on several criteria. These criteria include: the stage of maturity of the wheat crop, potential yield of the field, vulnerability of the wheat varieties, immediate weather conditions and long range forecast, and cost of treatment. In eastern and south central Nebraska, all of these criteria in 1982 pointed to a severe foliar disease outbreak; thus, growers were encouraged to have fields sprayed. Those growers that took this course of action benefited from the results.

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Table 1.--Chemical control of wheat leaf rust, septoria leaf blotch, and tan spot in 1982, Clay Center, Nebraska.

Treatment and rate (ai)/acre	Number of applications[1]	Disease index[2] Percentage of leaf rust	Disease index[2] Percentage of leaf spot[3]	Yield bu/A	1000-kernel wt(g)
Control		[4]100.0a	8.70abc	27.2c	28.6c
Dithane M-45 80WP 1.6 lb	1	66.0b	8.90a	32.1c	33.6ab
Bayleton 2EC 4 fl oz	1	58.0b	8.75ab	33.7bc	32.8abc
RH-5781F 1.5EC + Tween 20 0.4 lb	2	40.0c	8.35cd	40.7ab	34.0ab
Bayleton 50WP 2 oz	1	37.0cd	8.70abc	40.2ab	33.2abc
Dithane M-45 80WP 1.6 lb	2	36.0cd	8.70abc	31.3c	34.0ab
RH-5781F 1.5EC + Tween 20 0.2 lb	2	36.0cd	8.55abcd	34.1bc	32.8abc
Tilt (CGA-64250) 3.6EC 2.6 fl oz	1	27.0cde	8.40bcd	41.9a	31.2bc
M-8225F 1.6 lb	2	18.0def	8.75ab	33.7bc	33.2abc
RH-5781F 1.5EC + Dithane M-45 80W 0.4 lb + 1.6 lb	2	9.0ef	8.30d	42.8a	35.6abc
RH-5781F 1.5EC + Dithane M-45 80W 0.2 lb + 1.6 lb	2	4.2f	8.35cd	39.5ab	36.8a
Experimental mean		39.2	8.6	36.1	33.25

[1]Application dates were June 1 and June 7, which corresponded to growth stage 10.1 (midlate boot) and 10.5 (flowering) on the Feekes Large Scale of wheat plant development.

[2]Plant leaf rust is the severity on the flag leaf. The scale appraising leaf spot intensity is that developed by Saari and Prescott (1).

[3]Septoria leaf blotch (Septoria tritici) was the predominant leaf spot.

[4]Numbers in a column with a letter in common are not significantly different (DMRT P = 0.05).

WATKINS - SPEAKER

Q. Prestes: You mentioned that you make a year by year decision as to whether or not to spray. How do you decide when to treat?

A. We monitor the leaf spot disease ourselves because they're going to start early, and then we watch very closely the newsletters that come from Oklahoma and Kansas to see what is developing on leaf rust problems. If they're reporting severe leaf rust in those States (we get our leaf rust inoculum from those southern States) and we start seeing leaf rust in early June, and we've had some wet weather and the long range forecast is for wet weather then we begin getting geared up for recommending that the wheat be sprayed. But I've been in Nebraska 8 years now and there's been only 1 year out of 8 that I have really recommended fungicides be used on wheat. In most years, the disease does not move up that extensively on the flag leaf. Now we're getting more into irrigated continuous wheat, and then you do supply enough moisture to give quite a bit of leaf rust in areas where before you didn't see much leaf rust. In those situations, it may pay to routinely spray with fungicides.

Q. Dave Marshall: What fungicides do you use?

A. We used Dithane M-45 or Manzate 200.

Q. Does the $20 per acre refer to the total cost of application?

A. Yes, that's two applications. It varies with how much the aerial application costs.

Q. Why would the treatment reduce yield?

A. I can't explain that now. With some of the treatments that are experimental fungicides, you get some good yields. When you are dealing with farmers, you don't talk about the experimentals. They cannot use them. We feel if we would consistently show a 5-bushel or more increase we would probably recommend spraying.

Comment by Fehrmann: You have to consider the effects of the modern fungicides. They increase the yield much more, plus they are more expensive.

A. Right, but Mancozeb was the only fungicide we had registered for wheat up until last year. We got Bayleton registration last year. So, that's the only product our farmers had other than Zineb for rust.

Q. R. E. Gaunt: You had a fairly severe season and you got fairly good control of the leaf rust. Further, you had severe infections with septoria. Is it impractical to treat those diseases? I think that perhaps the reason you are not getting those

yield responses is because you are not applying your fungicide early enough.

A. We're going to try some fall treatments this fall and then go to spring treatments.

Q. Boyd: In some of your slides, the seedlings looked extremely yellow. What do you attribute that to?

A. In the spring, we get a lot of yellowed leaves. This may be due to the cool soils and nitrogen tie up. In that instance, I think the yellowing is due to infection by septoria. I don't think it is predisposed by nitrogen tie up. But we also have soilborne mosaic, and it was in that field too. So, there could be some interactions. I guess what I'm saying is, there could have been a lot of factors that cause that yellowing and we need to take a closer look. A lot of the farmers in Nebraska worry about their wheat because it's too wet to plant corn. If they had corn in the ground, they could forget their wheat. We get a lot of calls asking why is the wheat yellowing? One of the reasons is the heavy Septoria or tan spot infections.

CHEMICAL CONTROL OF SPECKLED LEAF BLOTCH OF WHEAT IN ISRAEL

O. Carmi, J. Eshel, and Z. Eyal[1]

Speckled leaf blotch of wheat incited by Septoria tritici Rob. ex Desm. (perfect state: Mycosphaerella graminicola [Fuckel] Schroeter) is a major wheat disease in many parts of the world, causing serious reductions in yield (4). Under severe epidemics, some vulnerable wheat cultivars may suffer 30 to 50% losses in yield, resulting in shriveled grain unfit for milling.

Fungicide protection is used in some leaf blotch affected countries as a stopgap measure to secure high yields of resistant-deficient cultivars, usually to deal with unexpected outbreaks (1, 2, 3, 5, 8, 9). In a few cases, chemical control of speckled leaf blotch is used as part of the crop management system implemented in controlling several foliar pathogens (1, 7).

The lengthy association between the pathogen and the wheat host in Israel and in the Mediterranean region during the growing season (November-April), and the drastic fluctuations in the amount, frequency, and distribution of rainfall impose difficulties in formulating an effective chemical control program within economic and consumer health considerations.

Chemical control programs of speckled leaf blotch use protectant fungicides (thiocarbamates) and, in a few cases, systemic fungicides are used singly or in combination with protectants.

In the present study, the following aspects of chemical control of speckled leaf blotch were evaluated: a) the affect of various fungicides on disease control - protectant (maneb) and systemic (benomyl, propiconazole, and triadimefon), and b) the effect of the number of and timing of fungicide applications on disease progress, severity, and yield parameters.

MATERIALS AND METHODS

The chemical control trials were conducted in two regions: the semiarid (300 to 350 mm of rainfall) southern coastal plains (Lakhish Experiment Station) and in the more rainy (500 to 600 mm of rainfall) central coastal plains region (Bet Dagan Experiment Station) over two growing seasons (1980-81 and 1981-82). Two commercial, susceptible spring wheat cultivars were sown in a paired-plot design: the early maturing dwarf (90 cm) cultivar 'Barkai' (Yt//Nrn 10/Bvr 21-1C/3/FA/Miriam 2), and the later maturing, semidwarf (112 cm) cultivar 'Lakhish' (Yt//Nrn 10/Bvr 21-1C/3/FA). The two cultivars were sown in paired microplots (2.5 x 12m) in a randomized block design. Each pair of cultivars was separated from the adjacent pair by 3-m fallowed borders. In addition, the effectiveness of the chemical control program was evaluated during the 1980-81 season in a commercial wheat field of the cultivar Barkai, located in the southern coastal plains (Saad), where the fungicides were applied by aircraft.

In the microplot trials, Septoria epidemics were established by weekly over-the-canopy spraying with a conidial suspension of virulent S. tritici isolate mixture. The artificial inoculations were initiated at the onset of tillering (GS 20) (10) and terminated prior to the emergence of the flag leaf (GS 36). The percent disease coverage was weekly assessed on 15 marked plants in each plot, starting prior to the emergence of the flag leaf minus one (GS 34). These plants were harvested separately at the end of the season for evaluation of yield components in addition to whole plot yield assessment. The chemical control program was initiated when pycnidia coverage of 5% was detected on flag leaf minus two (F-2), or on flag leaf minus three (F-3). These thresholds (moving action thresholds) were not very rigid, since the enforcement of the chemical control program was delayed if possible until the emergence of the flag leaf (GS 37-GS 40), the latest at the end of booting (GS 47). The fungicides were applied at the following rates: maneb at 2,000 g of active ingredient (a.i.) per hectare (ha) per application, benomyl (Benlate) at 400 g a.i./ha/application; propiconazole (Tilt EC 100) at 125 g. a.i./ha/application. The chemical control program was executed for the two cultivars simultaneously, using low volume/low pressure CO_2 operated back sprayers fitted with a boom.

RESULTS

The dwarf cultivar Barkai harbored significantly higher disease coverages and manifested a faster disease progress than Lakhish during the 2-year trials and locations. In the more rainy region (central coastal plains), the disease reached higher severity levels in both cultivars (figs. 1 and 2) than in the semiarid region (figs. 1, 2, 3, and 4). The most effective treatments in both seasons were those where two early successive (16 to 21-day intervals) propiconazole (Tilt) and benomyl (Benlate) fungicides were applied, resulting in slower disease progress, lower disease coverages, and significantly higher yields and kernel weights than the untreated inoculated controls (tables 1, 2, 3, and 4).

Single applications of propiconazole early in the season were more effective in controlling the disease and securing high yields, than those given after the recommended threshold, but less effective than the two successive early applications of that fungicide. Propiconazole applications where the threshold was misjudged (late applications) were still significantly more effective than the untreated inoculated controls and may be indicative of a certain degree of curative effect of propiconazole. Under high disease levels, this curative effect was less pronounced than under low-moderate disease levels. Despite low disease coverage resulting

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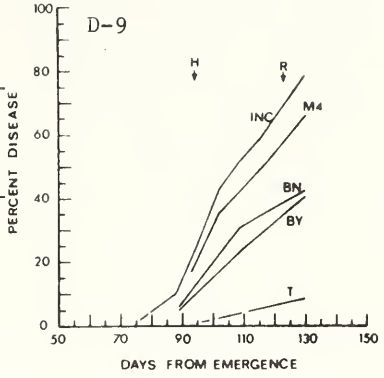
Figure 1.--The effect of protectant (maneb) and systemic (benomyl, propiconazole, and triadimefon) fungicides on Septoria leaf blotch progress and severity in the susceptible wheat cultivars 'Barkai' and 'Lakhish', Bet-Dagan Experiment Station, Israel, 1980-81.

Treatment	Disease severity percent[1]	Disease severity percent[2]
D-8 (Barkai)		
Inoculated control	81.9 c[3]	88.2 d
Maneb x 4	78.9 c	85.8 d
Bayleton x 2	48.7 b	68.0 c
Benlate x 2	43.5 b	57.2 b
Tilt x 2	33.2 a	46.3 a
D-9 (Lakhish)		
Inoculated control	58.3 e	78.7 d
Maneb x 4	49.6 d	65.8 c
Benlate x 2	30.9 c	42.5 b
Bayleton x 2	23.3 b	41.2 b
Tilt x 2	4.0 a	8.6 a

[1]Recorded 112 days from seedling emergence.
[2]Recorded 130 days from seedling emergence.
[3]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.



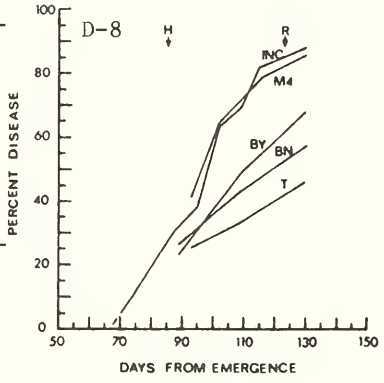
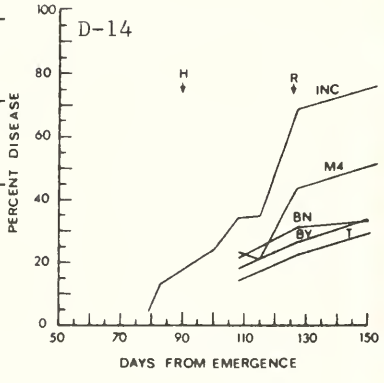


Figure 2.--Effect of protectant (maneb) and systemic (benomyl, propiconazole, and triadimefon) fungicides on Septoria leaf blotch progress and severity in the susceptible wheat cultivars 'Barkai' and 'Lakhish', Lakhish Experiment Station, Israel, 1980-81.

Treatment	Disease severity percent[1]	
	127 days	150 days
D-14 (Barkai)		
Inoculated control	68.7 c[2]	75.5 c
Maneb x 4	43.7 b	50.5 b
Benomyl x 2	24.9 ab	32.1 a
Bayleton x 2	29.4 ab	31.2 a
Tilt x 2	22.3 a	29.2 a
D-15 (Lakhish)		
Inoculated control	34.3 b	50.1 d
Maneb x 4	30.9 b	36.9 c
Benomyl x 2	17.2 a	29.1 bc
Tilt x 2	12.4 a	18.8 ab
Bayleton x 2	10.4 a	11.8 a

[1]Pycnidial coverage on the uppermost 3 leaves recorded 127 and 150 days after seedling emergence.
[2]Treatment means not followed by the same letter are significantly different at P =0.05 level as determined by Duncan's multiple range test.



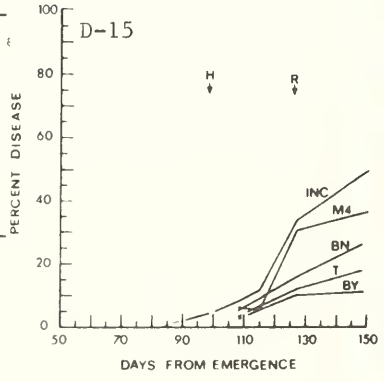
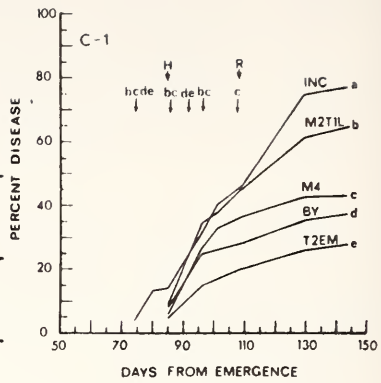
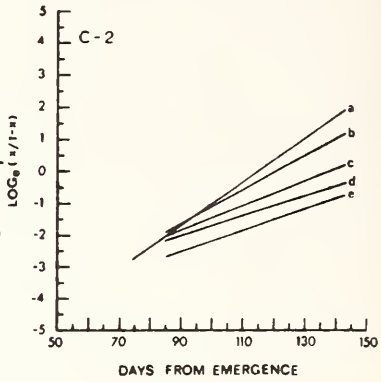


Figure 3.--Effect of fungicides, time, and number of applications on Septoria leaf blotch progress and severity (untransformed and transformed) in the susceptible, dwarf wheat cultivar 'Barkai', Lakhish Experiment Station, Israel, 1981-82.

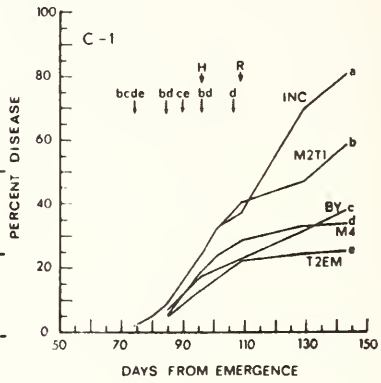


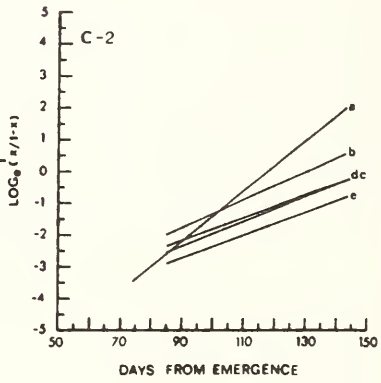


Treatment	Regression coefficient[1]	Disease severity percent[2] 109 days	129 days	143 days
C*				
A. Inoculated cont.	0.068	46.3 c[3]	74.9 d	77.3 d
B. Maneb x 2 + Tilt x 1 (L)	0.054	45.4 c	61.5 c	64.5 c
C. Maneb x 4	0.039	36.6 bc	42.9 b	43.4 b
D. Bayleton x 2	0.031	28.3 ab	35.4 ab	37.5 ab
E. Tilt x 2 (E+M)	0.034	20.2 a	25.9 a	27.9 a

[1]Regression coefficient of transformed pycnidial coverage ($Log_e(X/1-X)$).
[2]Pycnidial coverage of the uppermost 3 leaves recorded at 109, 129, and 143 days from emergence.
[3]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* C-1 and C-2, untransformed and transformed, respectively.
** H = heading date; R = last day of rain.

Figure 4.--The effect of fungicides, time, and number of applications on Septoria leaf blotch progress and severity (untransformed and transformed) in the susceptible, semidwarf wheat cultivar 'Lakhish', Lakhish Experiment Station, Israel, 1981-82.





Treatment	Regression coefficient[1]	Disease severity percent[2] 109 days	129 days	143 days
C*				
A. Inoculated cont.	0.079	37.6 bc[3]	70.0 c	81.3 d
B. Maneb x 2 + Tilt x 1 (L)	0.044	40.6 c	47.3 b	54.2 c
C. Bayleton x 2	0.038	23.1 a	31.7 a	38.3 b
D. Maneb x 4	0.036	28.8 ab	33.3 a	34.1 ab
E. Tilt x 2 (E+M)	0.034	22.5 a	24.7 a	25.5 a

[1]Regression coefficient of transformed pycnidial coverage ($Log_e(X/1-X)$).
[2]Pycnidial coverage of the uppermost 3 leaves recorded at 109, 129, and 143 days from emergence.
[3]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* C-1 and C-2, untransformed and transformed, respectively.
** H = heading date; R = last day of rain.

Table 1.--The effect of fungicides, timing, and number of applications on yield and kernel weight of the susceptible, dwarf wheat cultivar 'Barkai', in a chemical control trial of Septoria leaf blotch, Lakhish Experiment Station, Israel, 1980-81.

Treatment and number of applications	Yield[1]		Kernel weight		Disease severity percent[2]
	kg/h	Loss percent	g/1000	Loss percent	
Propiconazole X 1 (E)*	7297.9 a[3]	7.2	39.4 abc	5.7	21.1 b
Propiconazole X 1 (M)	6983.6 a	11.2	37.7 a	9.7	40.1 c
Propiconazole X 2 (E+M)	7869.6 a	+0.1	40.8 bc	2.2	22.4 b
Benomyl X 2 (E+M)	7451.2 a	5.2	37.8 a	9.4	24.9 bc
Triadimefon X 2 (E+M)	7758.3 a	1.3	38.7 ab	7.2	29.4 bc
Maneb X 2 (E+M)	7710.3 a	1.9	38.4 ab	8.1	56.9 de
Maneb X 3 (E+M+L)	7781.0 a	1.0	38.7 ab	7.4	26.1 bc
Maneb X 4 (E+M+L)	6587.9 a	16.2	37.9 a	8.9	43.7 cd
Protected control	7861.5 a	0.0	41.7 c	0.0	1.3 a
Inoculated control	6422.2 a	18.3	36.9 a	11.4	68.7 e

[1]Yield = kg/hectare, kernel weight = 1000 kernel weight (gm).
[2]Mean disease coverage of 3 uppermost leaves recorded 127 days after emergence GS 11.1(75-80)--Late milk stage.
[3]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* E = Early, M = Middle, and L = Late application.

Table 2.--The effect of fungicides, timing, and number of applications on yield and kernel weight of the susceptible, dwarf wheat cultivar 'Barkai', in a chemical control trial of Septoria leaf blotch, Bet-Dagan Experiment Station, Israel, 1980-81.

Treatment and number of applications	Yield[1]		Kernel weight		Disease severity percent[2]
	kg/h	Loss percent	g/1000	Loss percent	
Propiconazole X 1 (E)*	5348.0 b[3]	25.0	31.3 abcd	15.0	38.4 bc
Propiconazole X 1 (M)	5404.8 b	24.2	33.1 bcde	10.2	59.9 e
Propiconazole X 2 (E+M)	5964.6 b	16.4	34.2 cde	7.1	33.2 b
Benomyl X 2 (E+M)	5398.0 b	24.3	34.9 de	4.9	43.5 cd
Triadimefon X 2 (E+M)	5334.0 b	25.2	31.2 abcd	15.2	48.7 d
Maneb X 2 (E+M)	5438.7 b	23.7	29.8 abc	19.1	60.9 e
Maneb X 3 (E+M+L)	5007.8 b	29.8	29.7 abc	19.2	67.9 e
Maneb X 4 (E+M+L)	4891.9 b	31.4	29.2 ab	20.6	78.9 e
Protected control	7131.6 c	0.0	36.8 e	0.0	0.1 a
Inoculated control	3814.9 a	46.5	27.7 a	24.8	81.9 e

[1]Yield = kg/hectare, kernel weight = 1000 kernel weight (gm).
[2]Mean disease coverage of 3 uppermost leaves recorded 112 days after emergence GS 11.1-11.2 (75-83)--Late milk stage.
[3]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* E = Early, M = Middle, and L = Late application.

Table 3.--The effect of fungicides, timing, and number of applications on yield and kernel weight of the susceptible, dwarf wheat cultivar 'Barkai', in a chemical control trial of Septoria leaf blotch, Lakhish Experiment Station, Israel, 1981-82.

Treatment	Number of application and timing	Yield[1] kg/h	Yield[1] Loss percent	Kernel weight g/1000	Kernel weight Loss percent
Propiconazole	1 (E)*	7240.5 bc[2]	6.1	35.2 bc	12.2
Propiconazole	1 (M)	6793.9 abc	11.9	36.4 cde	9.1
Propiconazole	1 (L)	6323.7 ab	17.9	32.9 a	17.7
Propiconazole	2 (E+M)	7209.9 bc	6.5	39.2 g	2.2
Propiconazole	2 (M+L)	7077.9 abc	8.2	36.2 cd	9.7
Propiconazole	2 (E+L)	7261.4 bc	5.8	37.9 f	5.3
Triadimefon	2 (E+M)	7370.3 bc	4.4	36.8 def	8.1
Maneb	4 (E+M+L)	7384.6 bc	4.2	37.7 ef	5.9
Maneb + Propiconazole	2 + 1 (E+M+L)	7361.4 bc	4.5	37.9 f	5.3
Inoculated untreated	0	6070.5 a	21.3	34.1 ab	14.9
Protected control**	4 (E+M+L)	7711.3 c	0.0	40.0 g	0.0

[1]Yield = kg/hectar, kernel weight = 1000 kernel weight (gm).
[2]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* E = early, M = middle, and L = late application.
** Propiconazole (Tilt) EC 250.

Table 4.--The effect of fungicides, timing, and number of applications on yield and kernel weight of the susceptible, semidwarf wheat cultivar 'Lakhish', in a chemical control trial of Septoria leaf blotch, Lakhish Experiment Station, Israel, 1981-82.

Treatment	Number of application and timing	Yield[1] kg/h	Yield[1] Loss percent	Kernel weight g/1000	Kernel weight Loss percent
Propiconazole	1 (E)*	7407.4 ab[2]	10.9	39.2 ab	6.0
Propiconazole	1 (M)	7689.7 ab	7.5	39.1 a	6.2
Propiconazole	1 (L)	7894.8 ab	5.1	40.2 abcd	3.6
Propiconazole	2 (E+M)	7412.3 ab	10.9	40.5 bcde	2.9
Propiconazole	2 (M+L)	7972.8 ab	4.1	40.3 abcd	3.3
Propiconazole	2 (E+L)	7785.8 ab	6.4	40.9 cde	1.8
Triadimefon	2 (E+M)	7453.9 ab	10.4	39.8 abc	4.6
Maneb	4 (E+M+L)	7908.2 ab	4.9	40.5 bcde	2.9
Maneb + Propiconazole	2 + 1 (E+M+L)	7496.9 ab	9.8	41.4 de	0.9
Inoculated untreated	0	7160.8 a	13.9	39.5 ab	5.4
Protected control**	4 (E+M+L)	8315.9 b	0.0	41.7 e	0.0

[1]Yield = kg/hectar, kernel weight = 1000 kernel weight (gm).
[2]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.
* E = early, M = middle, and L = late application.
** Propiconazole (Tilt) EC 250.

from two successive triadimefon (Bayleton) applications, sometimes resulted in losses in yield greater than other fungicides.

The protectant fungicide maneb was found to be less effective than the systemics in controlling speckled leaf blotch during the 2-year trials, regardless of the number of applications. This is partly due to the fact that when the control program was initiated in the artificially inoculated microplots, the disease may have been amidst the latent period of the last inoculation (2 to 3 weeks prior to chemical control initiation). In addition, the fungicides were intentionally applied at the maximum intervals recommended, thus maneb was applied at 14-day intervals. A combination of two maneb applications followed by one propiconazole application, where the systemic intended to correct misjudgements in threshold selection, was as effective as the equivalent protectant treatment (figs. 3 and 4). The systemic fungicide did not provide the needed curative action.

In the trials conducted in a commercial field at Saad, the disease developed rather slowly on the susceptible cultivar Barkai due to long rainless intervals during the season. The chemical control was initiated when severity on F-2 reached 5% at the beginning of anthesis. One application of propiconazole resulted in 40.2% gains in yield from the untreated control (2967 kg/ha), and was significantly more effective than the other fungicides (table 5). A yield gain of 400 kg/ha (gain of 13.5%) economically justified the cost of chemicals and aircraft. This was equivalent to cost of one application of each of the systemic fungicides (benomyl, propiconazole, and triadimefon) and three applications of maneb at the time the trial was conducted.

DISCUSSION

Chemical control of speckled leaf blotch of wheat in Israel resulted in significantly higher yields and kernel weights and was economically justified. The systemic fungicides with curative properties and longer protective action against several foliar diseases of wheat were effective even when the action threshold was misjudged or when the chemical protection program was improperly executed. The dwarf cultivar Barkai required an earlier enforcement of the control program than the one implemented (5% coverage on F-2) and shortening the interval span. The action threshold implemented in the trials was suitable for the later, taller cultivar Lakhish under the experimental conditions. Chemical control programs of speckled leaf blotch should thus be adjusted to cultivar receptivity, disease progress, and vulnerability. It is obvious that chemical control of speckled leaf blotch will depend on disease surveying of each wheat field in disease prone areas. Disease surveying will ensure early detection, assess disease progress, and thus aid in making proper decisions to maximize the effectiveness of the disease control program.

The recently reported tolerance to carbendazim in Septoria nodorum (6) makes the combination of systemic and protectant fungicides more attractive. The application of protectant and systemic fungicide to follow did not provide the corrective action thought by the systemic fungicide. It is suggested that the sequence should be tried in reverse, namely successive protectant applications should follow one application of the systemic applied at the action threshold, or the two fungicides are mixed and applied jointly probably at a lower dose.

Table 5.--The effect of fungicides, timing, and number of applications on yield and kernel weight of the susceptible, dwarf wheat cultivar 'Barkai', in a chemical control trial of Septoria leaf blotch sprayed by aircraft, Saad, Israel, 1980-81.

Treatment	Number of application	Yield		Kernel weight	
		kg/h	Percent gain	g/1000	Percent gain
Propiconazole	2	3637 b[1]	22.6	39.3 bc	8.8
Propiconazole	1	4161 e	40.2	40.0 c	10.9
Benomyl	1	3513 b	18.4	39.3 bc	9.0
Triadimefon	1	3825 c	28.9	38.4 b	6.4
Maneb	3	3950 d	33.1	39.9 c	10.7
Untreated control	0	2967 a	0.0	36.1 a	0.0

[1]Treatment means not followed by the same letter are significantly different at P = 0.05 level as determined by Duncan's multiple range test.

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EYAL - SPEAKER

Q. Fried: When do the epidemics usually start in Israel?

A. Usually, it will start about a month or sometimes more than that before anthesis.

Q. P. M. Fried: What is the yield potential for your tolerant varieties?

A. Everyone will say the tolerant varieties are low yield or low kernel weight. All our varieties are in the range of 6 to 7 tons per hectare, very high yielding. So, we do not accept the argument about low potential yield. The yield level and maturity level are nearly the same as for the nontolerant varieties.

Q. Robert Hosford: A soil scientist found that stress before and during flowering, had the greatest effect on yield. I was wondering why you started analysis after anthesis instead of just a little bit before?

A. We are studying one parameter--kernel weight. We are mainly interested in working during the grain filling process.

Q. Ricardo Madariaga: Can you explain a little more the idea, the physiological process changes. What changes occur during drought, septoria and desiccation? Can you expand that idea?

A. The desiccant is working as a systemic toxin. I didn't say the desiccant physiologically is the same as disease. It is quite possible that they are operating on different physiological mechanisms. The septoria in this case, which is usually the major contributor, will affect photosynthesis and water relations.

SOURCES OF RESISTANCE TO SEPTORIA NODORUM IN BRAZIL

A. M. Prestes and J. M. Fernandes[1]

Septoria nodorum Berk., the imperfect stage of Leptosphaeria nodorum Müller, incites a disease in wheat known as glume blotch. The disease causes significant yield reductions each year in Brazil where warm, humid weather is frequently favorable for node, leaf, and head infections (3). Unfortunately, the weather in most of the wheat growing districts also favors the development of assorted other pathogens (4), including Helminthosporium sativum P. K. and B., Septoria tritici Rob and Desm., S. avenae f. sp. triticea T. Johnson, and Phoma spp. These organisms often cohabit the same host tissue and induce similar symptoms. In these events, the presence of one pathogen or disease is masked or confounded by the presence of another. Monitoring the incidence or importance of individual components of the complex is therefore difficult.

In evaluating breeding lines and selections for resistance to glume blotch, we augment field screening trials with an extensive greenhouse test. Seed of those lines judged most promising in open field culture is planted in large pots containing 7 kg of soil each. A typical test includes about 100 lines planted--each of eight pots. Upon emergence, seedlings are thinned to five uniform members per pot, and the plants are maintained in the greenhouse until heading. At anthesis, four pots of each line are placed in a walk-in humidity chamber and inoculated uniformly with S. nodorum. The remaining four pots of each line, serving as checks, are placed in a second chamber and misted with water.

Inoculum is prepared by increasing each of numerous isolates of the fungus on wheat flour agar under 24-hour days at 22°C. At 7 to 10 days of culture, when sporulation is generally profuse, the cultures are flooded with sterile water and brushed to dislodge pycnidiospores. The resulting suspensions are massed, adjusted to 10^6 spores/ml, and applied to plants as a mist by a DeVilbiss spray nozzle operated at 25 psi. Inoculated plants are maintained on 12-hour days for 48 hours, during which a mist of distilled water is administered for 60 seconds in every 15 minutes. This treatment suffices to assure continuous free water on foliar surfaces and 100% RH. At 48 hours, the plants are placed on greenhouse benches and maintained until maturity. Check plants are treated identically except that inoculum is replaced by sterile water.

Plants are examined at 10 days following inoculation and repeated at 10-day intervals thereafter until harvest. Incidence of nodal, foliar, and head (including rachis, glume, and awn) infections are recorded (2). Yield records include details on the various components (1).

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Results obtained from several tests carried out in the past three years identify the following wheat cultivars as showing the highest level of resistance to S. nodorum: Alvarez 110, Arthur (CI 14425), BH 1146, Br 6, Br 8, Hadden, Chinese/Aegilops umbelulata, CNT 1, Coker 762, Coker 76-35, Coxilha, Delta Queen, IAS 63, Lagoa Vermelha, Minuano 82, PAT 7219, PF 7576, PF 7815, Oasis, Toropi, and Transfer.

When tested under this system, Triticum spelta, T. durum, T. monococcum, and T. timopheevi are highly susceptible. On the other hand, of the 19 lines of Aegilops squarrosa tested, seven appear to be resistant. These lines are under continual observation and evaluation as possible parental material in breeding programs.

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PRESTES - SPEAKER

Q. Madariaga: You mentioned something about toxicity of aluminum that predisposes wheat to Septoria.

A. That is just a hypothesis I made. Acid soils are sometimes quite stressful. You lime the field and the lime just gets in a very narrow layer of the soil. As the roots go down, they reach the top level of aluminum and maybe this would predispose some of the plants to Septoria.

Q. Jim Frank: What weight do you put on the node infection? For example, if you had a certain cultivar that had an eight rating on the flag leaf and nothing on the node, how would you rate it versus another variety which had eight on the node? Which one would you select?

A. In general, we are most concerned with the heads and the nodes. In the fields, we inoculate right on the flag leaf. In the field, results we had inoculated about five or six times, and we repeat this many times in the evening about the same way Dr. Scharen does here except we don't put the plot under plastic, it is not necessary. We

do put the inoculum straight on the flag leaf, and we might use quite a lot of inoculum. It may be that the leaves are a more susceptible tissue, and this may be the reason that the infection on the nodes takes longer. It could be that you have to score a little bit later.

Q. Is the node infection very important?

A. Yes. Take, for example, the variety BH1146. Even though you get high infection on the flag leaf, you don't get much yield reduction and that more or less corresponds to the scoring on the heads and the nodes. These two get the most high infection level. With infection on the nodes and heads, however, you do get a negative yield response. So, we consider disease on the head more than on the nodes.

Q. Bob Tomerlin: Is lodging a particular problem? Is that why you're mainly concerned with infection on the nodes?

A. Yes, that is a problem, but I think they are independent things. You have increased lodging in the presence of infection by Septoria. You also have lodging because of wind and an excess of nitrogen, which will also cause lodging.

EVALUATION OF FUTURE NEEDS FOR COOPERATIVE WORK ON SEPTORIA DISEASES

Howard E. Waterworth[1]

There is little doubt that Septoria causes significant losses to wheat production. In the United States, various estimates have losses at about one percent of potential production. This may not seem like much, but it certainly is if we consider the value of the crop. The 1981 official Agricultural Statistics shows production at nearly 2.8 billion bushels. At a value of $3.66 per bushel, the value of that crop was worth approximately $10.2 billion. Therefore, losses cost American growers about $100 million. Not many diseases in this country are that costly.

By comparison, some of the single diseases of other major crops are (using 1981 dollar values) Verticillium wilt of cotton--loss $95 million; potato late blight--$71 million; and tobacco wildfire--$46 million. Only a few diseases, all of major crops, have a greater economic impact--among them cereal rusts and Phytophthora root rot of soybeans.

We've all seen loss data due to Septoria diseases from other countries where figures of 5, 20, and even 35 percent have been reported. So, I'm convinced that this disease deserves every bit of the current combined attention it receives on the part of the private sector, State Agricultural Experiment Stations, and the Federal Government.

I have some brief comments on your 1976 recommendations, from the proceedings for dealing with Septoria. These comments concern epidemiology and breeding for resistance, disease and loss assessment, uniformity of inoculation and disease rating procedures, and germplasm exchange.

You may not know it, but your recommendations to "administrators" among others have been taken to heart--at least in the ARS--or has it been coincidental? In any case, ARS planners agree with you for the most part, and this has been demonstrated by recent increased funding or expected increases in our Agency's plan for the next six years. For example, you suggested "breeding for resistance," we are expanding germplasm modification technology; you mention disease loss assessment, we identified as a problem that ARS should address the development of disease loss methods technology; and where you've emphasized germplasm exchange, ARS has expanded nearly all aspects of germplasm--collection, maintenance, evaluation, distribution, enhancement, and data systems--as many of you are aware. It seems to me the ARS has compiled rather well with your recommendations.

I don't know exactly how your research priority list of 1983--if you develop one--might compare with your 1976 list. I suspect it wouldn't differ a whole lot. It is very clear from the reports this week that, while progress has been made along several lines, much remains to be done, especially in getting effective tolerance into high yielding varieties. Dr. Jones mentioned a priority research need "an assessment of components to resistance." I was pleased to hear that because 2 months ago we approved a proposal by Dr. Kurt Leonard and a colleague in Israel to study "components to resistance in cereal crops."

Someone else mentioned, as a research priority, the understandings of physiological specialization of the pathogen and more on the genetics of resistance, though I'm not quite certain what that means. If it's the kind of biochemical approach that Dr. Caten is doing, I fully endorse it. I heard about "pyramiding" genes, too. This is good, but will it be possible without more basic work on the genes themselves?

I want to take this opportunity to mention very briefly some fairly new initiatives that ARS is undertaking in the way of long-term work on disease control. I think the following new approaches to solving disease problems apply to Septoria as well as to many other diseases.

I've already mentioned our increased germplasm activity. We are committed to an expansion and to the success of this effort for all commodities. Another thrust deals with new ways to manipulate and modify genes--of hosts, of pathogens, and of beneficial microorganisms. Some would call this genetic engineering technology development.

Induced resistance is another potentially useful new way to suppress disease that may have broad application. We already know it works in many host-pathogen combinations.

We need to know more about the biochemistry of host-pathogen interactions at the cellular level. What compounds are produced by each partner, when, why, and how much? With this information, we may be able to regulate these interactions. Indeed, "bioregulation" research is currently receiving considerable support in ARS. There is the question of cultivar mixtures or multilines as a means to suppress Septoria diseases. Dr. Jones raised a question about the potential of this approach. I understand the use of multilines was successful and widely adopted by farmers in Iowa as a way to suppress crown rust in oats. We recently approved a new project to study this approach to suppressing wheat rusts in Minnesota. We can only hope that this approach will contribute to our goals.

Furthermore, we have biocontrol with organisms. The entomologists have a 70-year lead on us pathologists in this approach to pest control. Only in the past 10 years have we pathologists really demonstrated the potential of this control measure. Yet, even today our biocontrol research program is far too small compared with the potential results I am convinced it will eventually offer. I note with interest reports by Fokkema, Flannigan, and others related to biocontrol of Septoria; so, there may well be potential here, too. ARS is also looking at the contributions that models will make in crop protection. Certainly, traditional breeding

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for resistance and use of fungicides will be our major defense against Septoria for a while, yet I have no doubt that some of these new approaches will impact on Septoria diseases in the very near future. Obviously, these approaches don't necessarily deal with the immediate disease problem out there now. From what I've heard this week, it seems to me you're on target. Many approaches are being pursued. It isn't necessary for me to review them now.

Not much has been said about the effect of changing cultural practices on Septoria in cereals. I am referring particularly to such things as multiple cropping, cropping under irrigation, and all kinds of conservation tillage practices. These and other practices are being rapidly adopted by growers. With conservation tillage, an increase in tan spot and Septoria diseases has already occurred in some places. A lot of questions need answers in this area, and we are expanding this kind of research.

You may be interested to know that ARS is planning a series of comprehensive workshops to identify priority research needs on selected major commodities. Soybeans, sugar crops, and wheat have been the first three commodities chosen for this analysis. I invite any comments you may have regarding ARS' wheat research program.

We are pleased that so many of our colleagues from overseas were able to be with us. We appreciated the opportunity to exchange data and ideas with you. We hope the trip was worth the effort for you. I would urge everyone to continue their efforts and to expand international cooperation as a means to expedite progress. This workshop contributed to that effort. It's up to each of you individually to carry on. I assure you that it is ARS' policy to share our resources with you--any item in our germplasm collections and any information in our libraries. Furthermore, our scientists are free to cooperate with foreign colleagues.

I also want to invite you to Beltsville, MD., especially our foreign colleagues. I will be pleased to assist you in any way.

Finally, on behalf of all of us, I thank you Al, Barry, and Joe, for your major roles in making this workshop successful. Thanks also to Dr. Jones for accepting the invitation and the excellent address.

WATERWORTH - SPEAKER

Q. Hosford: Is this transfer of germplasm from other grasses into wheat receiving any attention?

A. I don't know the answer to that. I'm sure there probably is work being done, but there are people on my staff who are in a better position to answer that and who probably will be trying it themselves.

Comment by Hosford: I'm curious because our group is quite interested in becoming involved in moving chromosomes from grasses to wheat where we take the resistance to a number of diseases and other factors.

Q. Masaad: Do you have a collection of varieties that have a high level of resistance to septoria in a particular country combined into one nursery?

A. I suspect that policies in the various countries would not allow such a nursery to come in.

Comment by Cunfer: I think he is saying there are several agencies now putting together nurseries of different kinds. His question is, is it feasible to have these combined in some way? I think we have the people here who could answer that.

Comment by Wilson: The Australian Septoria Nursery is mainly a domestic nursery and not designed for international use.

Comment by Prescott: CIMMYT could probably accept a small number of entries from cooperators for inclusion in the ISEPTON.

Comment by Al Scharen: I believe that in the near future programs such as the USDA's coordination of these nurseries will come under review, and whether this will fit into the priorities as far as ARS, USDA, is concerned is somewhat up in the air and questionable at this time. It is my feeling that USDA probably is going to become less involved with preparing and coordinating nurseries.

Comment by Ballantyne: I was wondering if perhaps we could have some working party to consider international nurseries.

Comment by Robin Wilson: I think the most important point is, if possible, that these resistant lines be available to other countries.

Comment by Sanderson: I think the question that emerges, there are two types of nurseries. In the United States, you've got nurseries in which germplasm is sent around for breeders to look at for possible use in their programs. In the western countries, you've got nurseries which contain breeders material which is sent to various locations to see how it performs.

Comment by Prescott: With regard to acceptance into the ISEPTON, the procedure is it must pass a test in Mexico at two septoria locations. Then, those that are low in septoria must pass tests for yield, quality, and resistance to other diseases.
Comment by Shaner: One of the problems is winter and spring wheat. We can grow both in Indiana, but we don't see a good spring wheat. We would probably need two nurseries, one of winter types and one of spring types.

Comment by Sanderson: Another problem with international nurseries is plant breeders rights.

Comment by Eyal: I was weighing the question given at the very beginning of the talk, and it's been disturbing me for quite awhile. I refer to the figure that was published in 1965 on the amount of

losses attributed to septoria. All of us would like to use this one percent, and it's been carried on for years in almost every publication that talks about losses and septoria. It's been very inflated; the figure has never been changed. I'm not certain what the figure is actually. The figure of one percent is a quarter of what the losses are attributed to septoria. The loss situation since 1965 has changed considerably. So, I think that we are using the figure very, very loosely. Of course, when we come to ask for money, we are quoting the one percent. I guess we should have quoted a higher percentage, but there is not enough data to figure a new one out. I think that it would be a very useful tool if the loss due to septorias could be separated from each other, one figure for S. nodorum and one for S. tritici. I think ARS could be the correct institute to develop a national assessment figure on losses due to septoria.

A. Waterworth: I couldn't possibly agree with you more. I doubt that anyone in this room would. Certainly, our technology to determine losses is very limited. It is not normally the role of ARS to determine crop losses, but if we assume that our role is to develop methods of determining losses, that may be the appropriate approach we need to take.

RESOLUTIONS

1. The participants of the International Workshop on Septoria Diseases of Cereals express their sincere thanks to A. L. Scharen for organizing the Workshop. The project has demanded a significant amount of time and effort. Through his efforts, the Workshop has been an important educational experience. The participants also extend their appreciation to Montana State University, to the members of the Department of Plant Pathology, to Joe Krupinsky for organizing the demonstration session, and to Barry Cunfer for serving as program chairman.

2. The participants of the International Workshop on Septoria Diseases of Cereals wish to acknowledge the important contribution of Eugene Sharp and the members of the Department of Plant Pathology at Montana State University for recognizing the problem of Septoria diseases of wheat in Montana and initiating research programs on their biology and control. This international workshop is a direct product of that work.

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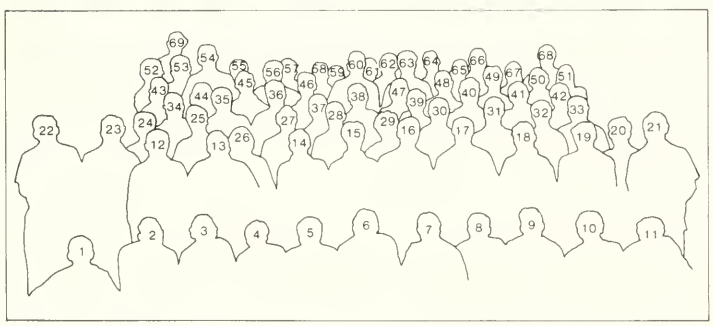

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